

99 Human Secreted Proteins

Related Applications

- 5 This application is a continuation-in-part of PCT/US02/09239, filed March 26, 2002, which in turn claims benefit of the following:

Application ::	Continuity Type::	Parent Application::	Parent Filing Date::
PCT/US02/09239	Continuation-in-part of	10/105,299	03/26/02
10/105,299	Non-provisional of	US 60/278,650	03/27/01
10/105,299	Continuation-in-part of	US 09/950,082	09/12/01
US 09/950,082	Non-provisional of	US 60/278,650	03/27/01
US 09/950,082	Continuation-in-part of	PCT/US00/06043	03/09/00
US00/06043	Non-provisional of	US 60/167,061	11/23/99
US00/06043	Non-provisional of	US 60/124,146	03/12/99
US 09/950,082	Continuation-in-part of	PCT/US00/06012	03/09/00
US00/06012	Non-provisional of	US 60/166,989	11/23/99
US00/06012	Non-provisional of	US 60/124,093	03/12/99
US 09/950,082	Continuation-in-part of	PCT/US00/06058	03/09/00
US00/06058	Non-provisional of	US 60/168,654	12/03/99
US00/06058	Non-provisional of	US 60/124,145	03/12/99
US 09/950,082	Continuation-in-part of	PCT/US00/06044	03/09/00
US00/06044	Non-provisional of	US 60/168,661	12/03/99
US00/06044	Non-provisional of	US 60/124,099	03/12/99
US 09/950,082	Continuation-in-part of	PCT/US00/06059	03/09/00
US00/06059	Non-provisional of	US 60/168,622	12/03/99
US00/06059	Non-provisional of	US 60/124,096	03/12/99
US 09/950,082	Continuation-in-part of	PCT/US00/06042	03/09/00
US00/06042	Non-provisional of	US 60/168,663	12/03/99
US00/06042	Non-provisional of	US 60/124,143	03/12/99
US 09/950,082	Continuation-in-part of	PCT/US00/06014	03/09/00
US00/06014	Non-provisional of	US 60/168,665	12/03/99
US00/06014	Non-provisional of	US 60/138,598	06/11/99
US00/06014	Non-provisional of	US 60/124,095	03/12/99
US 09/950,082	Continuation-in-part of	PCT/US00/06013	03/09/00
US00/06013	Non-provisional of	US 60/168,662	12/03/99
US00/06013	Non-provisional of	US 60/138,626	06/11/99
US00/06013	Non-provisional of	US 60/125,360	03/19/99
US 09/950,082	Continuation-in-part of	PCT/US00/06049	03/09/00
US00/06049	Non-provisional of	US 60/168,667	12/03/99
US00/06049	Non-provisional of	US 60/138,574	06/11/99
US00/06049	Non-provisional of	US 60/124,144	03/12/99
US 09/950,082	Continuation-in-part of	PCT/US00/06057	03/09/00
US00/06057	Non-provisional of	US 60/168,666	12/03/99
US00/06057	Non-provisional of	US 60/138,597	06/11/99
US00/06057	Non-provisional of	US 60/124,142	03/12/99
US 09/950,082	Continuation-in-part of	PCT/US00/06824	03/16/00

US00/06824	Non-provisional of	US 60/168,664	12/03/99
US00/06824	Non-provisional of	US 60/125,359	03/19/99
US 09/950,082	Continuation-in-part of	PCT/US00/06765	03/16/00
US00/06765	Non-provisional of	US 60/169,906	12/10/99
US00/06765	Non-provisional of	US 60/126,051	03/23/99
US 09/950,082	Continuation-in-part of	PCT/US00/06792	03/16/00
US00/06792	Non-provisional of	US 60/169,980	12/10/99
US00/06792	Non-provisional of	US 60/125,362	03/19/99
US 09/950,082	Continuation-in-part of	PCT/US00/06830	03/16/00
US00/06830	Non-provisional of	US 60/169,910	12/10/99
US00/06830	Non-provisional of	US 60/125,361	03/19/99
US 09/950,082	Continuation-in-part of	PCT/US00/06782	03/16/00
US00/06782	Non-provisional of	US 60/169,936	12/10/99
US00/06782	Non-provisional of	US 60/125,812	03/23/99
US 09/950,082	Continuation-in-part of	PCT/US00/06822	03/16/00
US00/06822	Non-provisional of	US 60/169,916	12/10/99
US00/06822	Non-provisional of	US 60/126,054	03/23/99
US 09/950,082	Continuation-in-part of	PCT/US00/06791	03/16/00
US00/06791	Non-provisional of	US 60/169,946	12/10/99
US00/06791	Non-provisional of	US 60/125,815	03/23/99
US 09/950,082	Continuation-in-part of	PCT/US00/06828	03/16/00
US00/06828	Non-provisional of	US 60/169,616	12/08/99
US00/06828	Non-provisional of	US 60/125,358	03/19/99
US 09/950,082	Continuation-in-part of	PCT/US00/06823	03/16/00
US00/06823	Non-provisional of	US 60/169,623	12/08/99
US00/06823	Non-provisional of	US 60/125,364	03/19/99
US 09/950,082	Continuation-in-part of	PCT/US00/06781	03/16/00
US00/06781	Non-provisional of	US 60/169,617	12/08/99
US00/06781	Non-provisional of	US 60/125,363	03/19/99
US 09/950,082	Continuation-in-part of	PCT/US00/07505	03/22/00
US00/07505	Non-provisional of	US 60/172,410	12/17/99
US00/07505	Non-provisional of	US 60/126,502	03/26/99
US 09/950,082	Continuation-in-part of	PCT/US00/07440	03/22/00
US00/07440	Non-provisional of	US 60/172,409	12/17/99
US00/07440	Non-provisional of	US 60/126,503	03/26/99
US 09/950,082	Continuation-in-part of	PCT/US00/07506	03/22/00
US00/07506	Non-provisional of	US 60/172,412	12/17/99
US00/07506	Non-provisional of	US 60/126,505	03/26/99
US 09/950,082	Continuation-in-part of	PCT/US00/07507	03/22/00
US00/07507	Non-provisional of	US 60/172,408	12/17/99
US00/07507	Non-provisional of	US 60/126,594	03/26/99
US 09/950,082	Continuation-in-part of	PCT/US00/07535	03/22/00
US00/07535	Non-provisional of	US 60/172,413	12/17/99
US00/07535	Non-provisional of	US 60/126,511	03/26/99
US 09/950,082	Continuation-in-part of	PCT/US00/07525	03/22/00
US00/07525	Non-provisional of	US 60/171,549	12/22/99
US00/07525	Non-provisional of	US 60/126,595	03/26/99
US 09/950,082	Continuation-in-part of	PCT/US00/07534	03/22/00
US00/07534	Non-provisional of	US 60/171,504	12/22/99
US00/07534	Non-provisional of	US 60/126,598	03/26/99

US 09/950,082	Continuation-in-part of	PCT/US00/07483	03/22/00
US00/07483	Non-provisional of	US 60/171,552	12/22/99
US00/07483	Non-provisional of	US 60/126,596	03/26/99
US 09/950,082	Continuation-in-part of	PCT/US00/07526	03/22/00
US00/07526	Non-provisional of	US 60/171,550	12/22/99
US00/07526	Non-provisional of	US 60/126,600	03/26/99
US 09/950,082	Continuation-in-part of	PCT/US00/07527	03/22/00
US00/07527	Non-provisional of	US 60/171,551	12/22/99
US00/07527	Non-provisional of	US 60/126,501	03/26/99
US 09/950,082	Continuation-in-part of	PCT/US00/07661	03/23/00
US00/07661	Non-provisional of	US 60/174,847	01/07/00
US00/07661	Non-provisional of	US 60/126,504	03/26/99
US 09/950,082	Continuation-in-part of	PCT/US00/07579	03/23/00
US00/07579	Non-provisional of	US 60/174,853	01/07/00
US00/07579	Non-provisional of	US 60/126,509	03/26/99
US 09/950,082	Continuation-in-part of	PCT/US00/07723	03/23/00
US00/07723	Non-provisional of	US 60/242,710	10/25/00
US00/07723	Non-provisional of	US 60/174,852	01/07/00
US00/07723	Non-provisional of	US 60/126,506	03/26/99
US 09/950,082	Continuation-in-part of	PCT/US00/07724	03/23/00
US00/07724	Non-provisional of	US 60/174,850	01/07/00
US00/07724	Non-provisional of	US 60/126,510	03/26/99
US 09/950,082	Continuation-in-part of	PCT/US00/14929	06/01/00
US00/14929	Non-provisional of	US 60/174,851	01/07/00
US00/14929	Non-provisional of	US 60/138,573	06/11/99
US 09/950,082	Continuation-in-part of	PCT/US00/07722	03/23/00
US00/07722	Non-provisional of	US 60/174,871	01/07/00
US00/07722	Non-provisional of	US 60/126,508	03/26/99
US 09/950,082	Continuation-in-part of	PCT/US00/07578	03/23/00
US00/07578	Non-provisional of	US 60/174,872	01/07/00
US00/07578	Non-provisional of	US 60/126,507	03/26/99
US 09/950,082	Continuation-in-part of	PCT/US00/07726	03/23/00
US00/07726	Non-provisional of	US 60/174,877	01/07/00
US00/07726	Non-provisional of	US 60/126,597	03/26/99
US 09/950,082	Continuation-in-part of	PCT/US00/07677	03/23/00
US00/07677	Non-provisional of	US 60/176,064	01/14/00
US00/07677	Non-provisional of	US 60/154,373	09/17/99
US00/07677	Non-provisional of	US 60/126,601	03/26/99
US 09/950,082	Continuation-in-part of	PCT/US00/07725	03/23/00
US00/07725	Non-provisional of	US 60/176,063	01/14/00
US00/07725	Non-provisional of	US 60/126,602	03/26/99
US 09/950,082	Continuation-in-part of	PCT/US00/09070	04/06/00
US00/09070	Non-provisional of	US 60/176,052	01/14/00
US00/09070	Non-provisional of	US 60/128,695	04/09/99
US 09/950,082	Continuation-in-part of	PCT/US00/08982	04/06/00
US00/08982	Non-provisional of	US 60/176,069	01/14/00
US00/08982	Non-provisional of	US 60/128,696	04/09/99
US 09/950,082	Continuation-in-part of	PCT/US00/08983	04/06/00
US00/08983	Non-provisional of	US 60/176,068	01/14/00
US00/08983	Non-provisional of	US 60/128,703	04/09/99

US 09/950,082	Continuation-in-part of	PCT/US00/09067	04/06/00
US00/09067	Non-provisional of	US 60/176,929	01/20/00
US00/09067	Non-provisional of	US 60/128,697	04/09/99
US 09/950,082	Continuation-in-part of	PCT/US00/09066	04/06/00
US00/09066	Non-provisional of	US 60/176,926	01/20/00
US00/09066	Non-provisional of	US 60/128,698	04/09/99
US 09/950,082	Continuation-in-part of	PCT/US00/09068	04/06/00
US00/09068	Non-provisional of	US 60/177,050	01/20/00
US00/09068	Non-provisional of	US 60/128,699	04/09/99
US 09/950,082	Continuation-in-part of	PCT/US00/08981	04/06/00
US00/08981	Non-provisional of	US 60/177,166	01/20/00
US00/08981	Non-provisional of	US 60/128,701	04/09/99
US 09/950,082	Continuation-in-part of	PCT/US00/08980	04/06/00
US00/08980	Non-provisional of	US 60/176,930	01/20/00
US00/08980	Non-provisional of	US 60/128,700	04/09/99
US 09/950,082	Continuation-in-part of	PCT/US00/09071	04/06/00
US00/09071	Non-provisional of	US 60/176,931	01/20/00
US00/09071	Non-provisional of	US 60/128,694	04/09/99
US 09/950,082	Continuation-in-part of	PCT/US00/09069	04/06/00
US00/09069	Non-provisional of	US 60/177,049	01/20/00
US00/09069	Non-provisional of	US 60/128,702	04/09/99
US 09/950,082	Continuation-in-part of	PCT/US00/15136	06/01/00
US00/15136	Non-provisional of	US 60/138,629	06/11/99
US 09/950,082	Continuation-in-part of	PCT/US00/14926	06/01/00
US00/14926	Non-provisional of	US 60/138,628	06/11/99
US 09/950,082	Continuation-in-part of	PCT/US00/14963	06/01/00
US00/14963	Non-provisional of	US 60/138,631	06/11/99
US 09/950,082	Continuation-in-part of	PCT/US00/15135	06/01/00
US00/15135	Non-provisional of	US 60/138,632	06/11/99
US 09/950,082	Continuation-in-part of	PCT/US00/14934	06/01/00
US00/14934	Non-provisional of	US 60/138,599	06/11/99
US 09/950,082	Continuation-in-part of	PCT/US00/14933	06/01/00
US00/14933	Non-provisional of	US 60/138,572	06/11/99
US 09/950,082	Continuation-in-part of	PCT/US00/15137	06/01/00
US00/15137	Non-provisional of	US 60/138,625	06/11/99
US 09/950,082	Continuation-in-part of	PCT/US00/14928	06/01/00
US00/14928	Non-provisional of	US 60/138,633	06/11/99
US 09/950,082	Continuation-in-part of	PCT/US00/14973	06/01/00
US00/14973	Non-provisional of	US 60/138,630	06/11/99
US 09/950,082	Continuation-in-part of	PCT/US00/14964	06/01/00
US00/14964	Non-provisional of	US 60/138,627	06/11/99
US 09/950,082	Continuation-in-part of	PCT/US00/26376	09/26/00
US00/26376	Non-provisional of	US 60/155,808	09/27/99
US 09/950,082	Continuation-in-part of	PCT/US00/26371	09/26/00
US00/26371	Non-provisional of	US 60/155,804	09/27/99
US 09/950,082	Continuation-in-part of	PCT/US00/26324	09/26/00
US00/26324	Non-provisional of	US 60/155,807	09/27/99
US 09/950,082	Continuation-in-part of	PCT/US00/26323	09/26/00
US00/26323	Non-provisional of	US 60/155,805	09/27/99
US 09/950,082	Continuation-in-part of	PCT/US00/26337	09/26/00

US00/26337	Non-provisional of	US 60/155,806	09/27/99
US 09/950,082	Continuation-in-part of	US01/13318	04/27/01
US01/13318	Non-provisional of	US 60/212,142	06/16/00
US01/13318	Non-provisional of	US 60/201,194	05/02/00
10/105,299	Continuation-in-part of	US 09/950,083	09/12/01
US 09/950,083	Non-provisional of	US 60/278,650	03/27/01
US 09/950,083	Continuation-in-part of	PCT/US00/06043	03/09/00
US00/06043	Non-provisional of	US 60/167,061	11/23/99
US00/06043	Non-provisional of	US 60/124,146	03/12/99
US 09/950,083	Continuation-in-part of	PCT/US00/06012	03/09/00
US00/06012	Non-provisional of	US 60/166,989	11/23/99
US00/06012	Non-provisional of	US 60/124,093	03/12/99
US 09/950,083	Continuation-in-part of	PCT/US00/06058	03/09/00
US00/06058	Non-provisional of	US 60/168,654	12/03/99
US00/06058	Non-provisional of	US 60/124,145	03/12/99
US 09/950,083	Continuation-in-part of	PCT/US00/06044	03/09/00
US00/06044	Non-provisional of	US 60/168,661	12/03/99
US00/06044	Non-provisional of	US 60/124,099	03/12/99
US 09/950,083	Continuation-in-part of	PCT/US00/06059	03/09/00
US00/06059	Non-provisional of	US 60/168,622	12/03/99
US00/06059	Non-provisional of	US 60/124,096	03/12/99
US 09/950,083	Continuation-in-part of	PCT/US00/06042	03/09/00
US00/06042	Non-provisional of	US 60/168,663	12/03/99
US00/06042	Non-provisional of	US 60/124,143	03/12/99
US 09/950,083	Continuation-in-part of	PCT/US00/06014	03/09/00
US00/06014	Non-provisional of	US 60/168,665	12/03/99
US00/06014	Non-provisional of	US 60/138,598	06/11/99
US00/06014	Non-provisional of	US 60/124,095	03/12/99
US 09/950,083	Continuation-in-part of	PCT/US00/06013	03/09/00
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US00/06013	Non-provisional of	US 60/138,626	06/11/99
US00/06013	Non-provisional of	US 60/125,360	03/19/99
US 09/950,083	Continuation-in-part of	PCT/US00/06049	03/09/00
US00/06049	Non-provisional of	US 60/168,667	12/03/99
US00/06049	Non-provisional of	US 60/138,574	06/11/99
US00/06049	Non-provisional of	US 60/124,144	03/12/99
US 09/950,083	Continuation-in-part of	PCT/US00/06057	03/09/00
US00/06057	Non-provisional of	US 60/168,666	12/03/99
US00/06057	Non-provisional of	US 60/138,597	06/11/99
US00/06057	Non-provisional of	US 60/124,142	03/12/99
US 09/950,083	Continuation-in-part of	PCT/US00/06824	03/16/00
US00/06824	Non-provisional of	US 60/168,664	12/03/99
US00/06824	Non-provisional of	US 60/125,359	03/19/99
US 09/950,083	Continuation-in-part of	PCT/US00/06765	03/16/00
US00/06765	Non-provisional of	US 60/169,906	12/10/99
US00/06765	Non-provisional of	US 60/126,051	03/23/99
US 09/950,083	Continuation-in-part of	PCT/US00/06792	03/16/00
US00/06792	Non-provisional of	US 60/169,980	12/10/99
US00/06792	Non-provisional of	US 60/125,362	03/19/99
US 09/950,083	Continuation-in-part of	PCT/US00/06830	03/16/00

US00/06830	Non-provisional of	US 60/169,910	12/10/99
US00/06830	Non-provisional of	US 60/125,361	03/19/99
US 09/950,083	Continuation-in-part of	PCT/US00/06782	03/16/00
US00/06782	Non-provisional of	US 60/169,936	12/10/99
US00/06782	Non-provisional of	US 60/125,812	03/23/99
US 09/950,083	Continuation-in-part of	PCT/US00/06822	03/16/00
US00/06822	Non-provisional of	US 60/169,916	12/10/99
US00/06822	Non-provisional of	US 60/126,054	03/23/99
US 09/950,083	Continuation-in-part of	PCT/US00/06791	03/16/00
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US00/06791	Non-provisional of	US 60/125,815	03/23/99
US 09/950,083	Continuation-in-part of	PCT/US00/06828	03/16/00
US00/06828	Non-provisional of	US 60/169,616	12/08/99
US00/06828	Non-provisional of	US 60/125,358	03/19/99
US 09/950,083	Continuation-in-part of	PCT/US00/06823	03/16/00
US00/06823	Non-provisional of	US 60/169,623	12/08/99
US00/06823	Non-provisional of	US 60/125,364	03/19/99
US 09/950,083	Continuation-in-part of	PCT/US00/06781	03/16/00
US00/06781	Non-provisional of	US 60/169,617	12/08/99
US00/06781	Non-provisional of	US 60/125,363	03/19/99
US 09/950,083	Continuation-in-part of	PCT/US00/07505	03/22/00
US00/07505	Non-provisional of	US 60/172,410	12/17/99
US00/07505	Non-provisional of	US 60/126,502	03/26/99
US 09/950,083	Continuation-in-part of	PCT/US00/07440	03/22/00
US00/07440	Non-provisional of	US 60/172,409	12/17/99
US00/07440	Non-provisional of	US 60/126,503	03/26/99
US 09/950,083	Continuation-in-part of	PCT/US00/07506	03/22/00
US00/07506	Non-provisional of	US 60/172,412	12/17/99
US00/07506	Non-provisional of	US 60/126,505	03/26/99
US 09/950,083	Continuation-in-part of	PCT/US00/07507	03/22/00
US00/07507	Non-provisional of	US 60/172,408	12/17/99
US00/07507	Non-provisional of	US 60/126,594	03/26/99
US 09/950,083	Continuation-in-part of	PCT/US00/07535	03/22/00
US00/07535	Non-provisional of	US 60/172,413	12/17/99
US00/07535	Non-provisional of	US 60/126,511	03/26/99
US 09/950,083	Continuation-in-part of	PCT/US00/07525	03/22/00
US00/07525	Non-provisional of	US 60/171,549	12/22/99
US00/07525	Non-provisional of	US 60/126,595	03/26/99
US 09/950,083	Continuation-in-part of	PCT/US00/07534	03/22/00
US00/07534	Non-provisional of	US 60/171,504	12/22/99
US00/07534	Non-provisional of	US 60/126,598	03/26/99
US 09/950,083	Continuation-in-part of	PCT/US00/07483	03/22/00
US00/07483	Non-provisional of	US 60/171,552	12/22/99
US00/07483	Non-provisional of	US 60/126,596	03/26/99
US 09/950,083	Continuation-in-part of	PCT/US00/07526	03/22/00
US00/07526	Non-provisional of	US 60/171,550	12/22/99
US00/07526	Non-provisional of	US 60/126,600	03/26/99
US 09/950,083	Continuation-in-part of	PCT/US00/07527	03/22/00
US00/07527	Non-provisional of	US 60/171,551	12/22/99
US00/07527	Non-provisional of	US 60/126,501	03/26/99

US 09/950,083	Continuation-in-part of	PCT/US00/07661	03/23/00
US00/07661	Non-provisional of	US 60/174,847	01/07/00
US00/07661	Non-provisional of	US 60/126,504	03/26/99
US 09/950,083	Continuation-in-part of	PCT/US00/07579	03/23/00
US00/07579	Non-provisional of	US 60/174,853	01/07/00
US00/07579	Non-provisional of	US 60/126,509	03/26/99
US 09/950,083	Continuation-in-part of	PCT/US00/07723	03/23/00
US00/07723	Non-provisional of	US 60/242,710	10/25/00
US00/07723	Non-provisional of	US 60/174,852	01/07/00
US00/07723	Non-provisional of	US 60/126,506	03/26/99
US 09/950,083	Continuation-in-part of	PCT/US00/07724	03/23/00
US00/07724	Non-provisional of	US 60/174,850	01/07/00
US00/07724	Non-provisional of	US 60/126,510	03/26/99
US 09/950,083	Continuation-in-part of	PCT/US00/14929	06/01/00
US00/14929	Non-provisional of	US 60/174,851	01/07/00
US00/14929	Non-provisional of	US 60/138,573	06/11/99
US 09/950,083	Continuation-in-part of	PCT/US00/07722	03/23/00
US00/07722	Non-provisional of	US 60/174,871	01/07/00
US00/07722	Non-provisional of	US 60/126,508	03/26/99
US 09/950,083	Continuation-in-part of	PCT/US00/07578	03/23/00
US00/07578	Non-provisional of	US 60/174,872	01/07/00
US00/07578	Non-provisional of	US 60/126,507	03/26/99
US 09/950,083	Continuation-in-part of	PCT/US00/07726	03/23/00
US00/07726	Non-provisional of	US 60/174,877	01/07/00
US00/07726	Non-provisional of	US 60/126,597	03/26/99
US 09/950,083	Continuation-in-part of	PCT/US00/07677	03/23/00
US00/07677	Non-provisional of	US 60/176,064	01/14/00
US00/07677	Non-provisional of	US 60/154,373	09/17/99
US00/07677	Non-provisional of	US 60/126,601	03/26/99
US 09/950,083	Continuation-in-part of	PCT/US00/07725	03/23/00
US00/07725	Non-provisional of	US 60/176,063	01/14/00
US00/07725	Non-provisional of	US 60/126,602	03/26/99
US 09/950,083	Continuation-in-part of	PCT/US00/09070	04/06/00
US00/09070	Non-provisional of	US 60/176,052	01/14/00
US00/09070	Non-provisional of	US 60/128,695	04/09/99
US 09/950,083	Continuation-in-part of	PCT/US00/08982	04/06/00
US00/08982	Non-provisional of	US 60/176,069	01/14/00
US00/08982	Non-provisional of	US 60/128,696	04/09/99
US 09/950,083	Continuation-in-part of	PCT/US00/08983	04/06/00
US00/08983	Non-provisional of	US 60/176,068	01/14/00
US00/08983	Non-provisional of	US 60/128,703	04/09/99
US 09/950,083	Continuation-in-part of	PCT/US00/09067	04/06/00
US00/09067	Non-provisional of	US 60/176,929	01/20/00
US00/09067	Non-provisional of	US 60/128,697	04/09/99
US 09/950,083	Continuation-in-part of	PCT/US00/09066	04/06/00
US00/09066	Non-provisional of	US 60/176,926	01/20/00
US00/09066	Non-provisional of	US 60/128,698	04/09/99
US 09/950,083	Continuation-in-part of	PCT/US00/09068	04/06/00
US00/09068	Non-provisional of	US 60/177,050	01/20/00
US00/09068	Non-provisional of	US 60/128,699	04/09/99

US 09/950,083	Continuation-in-part of	PCT/US00/08981	04/06/00
US00/08981	Non-provisional of	US 60/177,166	01/20/00
US00/08981	Non-provisional of	US 60/128,701	04/09/99
US 09/950,083	Continuation-in-part of	PCT/US00/08980	04/06/00
US00/08980	Non-provisional of	US 60/176,930	01/20/00
US00/08980	Non-provisional of	US 60/128,700	04/09/99
US 09/950,083	Continuation-in-part of	PCT/US00/09071	04/06/00
US00/09071	Non-provisional of	US 60/176,931	01/20/00
US00/09071	Non-provisional of	US 60/128,694	04/09/99
US 09/950,083	Continuation-in-part of	PCT/US00/09069	04/06/00
US00/09069	Non-provisional of	US 60/177,049	01/20/00
US00/09069	Non-provisional of	US 60/128,702	04/09/99
US 09/950,083	Continuation-in-part of	PCT/US00/15136	06/01/00
US00/15136	Non-provisional of	US 60/138,629	06/11/99
US 09/950,083	Continuation-in-part of	PCT/US00/14926	06/01/00
US00/14926	Non-provisional of	US 60/138,628	06/11/99
US 09/950,083	Continuation-in-part of	PCT/US00/14963	06/01/00
US00/14963	Non-provisional of	US 60/138,631	06/11/99
US 09/950,083	Continuation-in-part of	PCT/US00/15135	06/01/00
US00/15135	Non-provisional of	US 60/138,632	06/11/99
US 09/950,083	Continuation-in-part of	PCT/US00/14934	06/01/00
US00/14934	Non-provisional of	US 60/138,599	06/11/99
US 09/950,083	Continuation-in-part of	PCT/US00/14933	06/01/00
US00/14933	Non-provisional of	US 60/138,572	06/11/99
US 09/950,083	Continuation-in-part of	PCT/US00/15137	06/01/00
US00/15137	Non-provisional of	US 60/138,625	06/11/99
US 09/950,083	Continuation-in-part of	PCT/US00/14928	06/01/00
US00/14928	Non-provisional of	US 60/138,633	06/11/99
US 09/950,083	Continuation-in-part of	PCT/US00/14973	06/01/00
US00/14973	Non-provisional of	US 60/138,630	06/11/99
US 09/950,083	Continuation-in-part of	PCT/US00/14964	06/01/00
US00/14964	Non-provisional of	US 60/138,627	06/11/99
US 09/950,083	Continuation-in-part of	PCT/US00/26376	09/26/00
US00/26376	Non-provisional of	US 60/155,808	09/27/99
US 09/950,083	Continuation-in-part of	PCT/US00/26371	09/26/00
US00/26371	Non-provisional of	US 60/155,804	09/27/99
US 09/950,083	Continuation-in-part of	PCT/US00/26324	09/26/00
US00/26324	Non-provisional of	US 60/155,807	09/27/99
US 09/950,083	Continuation-in-part of	PCT/US00/26323	09/26/00
US00/26323	Non-provisional of	US 60/155,805	09/27/99
US 09/950,083	Continuation-in-part of	PCT/US00/26337	09/26/00
US00/26337	Non-provisional of	US 60/155,806	09/27/99
US 09/950,083	Continuation-in-part of	US01/13318	04/27/01
US01/13318	Non-provisional of	US 60/212,142	06/16/00
US01/13318	Non-provisional of	US 60/201,194	05/02/00

; wherein each of the above applications are all herein incorporated by reference in their entirety.

Field of the Invention

The present invention relates to human secreted proteins/polypeptides, and isolated nucleic acid molecules encoding said proteins/polypeptides, useful for detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating allergic and/or asthmatic diseases and disorders. Antibodies that bind these polypeptides are also encompassed by the present invention. Also encompassed by the invention are vectors, host cells, and recombinant and synthetic methods for producing said polynucleotides, polypeptides, and/or antibodies. The invention further encompasses screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further encompasses methods and compositions for inhibiting or enhancing the production and function of the polypeptides of the present invention.

Background of the Invention

The immune system is an intricate network of cells, tissues and soluble molecules that function to protect the body from invasion by foreign substances and pathogens. The major cells of the immune system are lymphocytes, including B cells and T cells, and myeloid cells, including basophils, eosinophils, neutrophils, mast cells, monocytes, macrophages and dendritic cells. In addition to these cellular components of the immune system, soluble molecules- such as antibodies, complement proteins, and cytokines- circulate in lymph and blood plasma, and play important roles in immunity.

The immune system can be subdivided into the acquired and innate immune systems. The cells of the innate immune system (e.g., neutrophils, eosinophils, basophils, mast cells) are not antigen specific and their action is not enhanced by repeated exposure to the same antigen. The cells of the acquired immune system (B and T cells) are antigen specific. Repeated exposure of B and T cells to an antigen results in improved immune responses (memory responses) produced by these cell types. The cells and products of the acquired immune system can recruit components of the innate system to mount a focused immune response. For a more extensive review of the immune system, see Fundamental Immunology, 4th edition, Ed. William Paul, Lippincott-Raven Pub. (1998).

An immune response is seldom carried out by a single cell type, but rather requires the coordinated efforts of several cell types. In order to coordinate an immune response, it is necessary that cells of the immune system communicate with each other and with other cells of the body. Communication between cells may be made by cell-cell contact, between membrane bound molecules on each cell, or by the interaction of soluble components of the immune system with cellular receptors. Signaling between cell types may have one or more of a variety of

consequences, including activation, proliferation, differentiation, and apoptosis. Activation and differentiation of immune cells may result in the expression or secretion of polypeptides, or other molecules, which in turn affect the function of other cells and/or molecules of the immune system.

5 The genes and proteins associated with this coordinated immune response are essential for the proper regulation and functioning of the immune system. Dysregulation of immune system-related genes and proteins may result in a variety of diseases and/or disorders, including immediate hypersensitivity diseases. Immediate hypersensitivity diseases, such as asthma, hay fever, and allergic conjunctivitis, are characterized by similar physiological mechanisms and generally are initiated by environmental antigens (e.g. pollen, dust, or molds). Patients suffering from the effects of these disorders are predisposed to react to specific external antigens. When these antigens contact certain tissues, such as ocular, nasal, or lung tissues, those tissues initiate an immune response and produce undesirable and frequently life-threatening symptoms. Over 35 million Americans suffer from allergic disorders, such as seasonal allergic rhinitis (hay fever), and asthma affects about 10 million Americans. These conditions are not only becoming more common but also more serious, with more people being hospitalized.

Molecules that stimulate or suppress immune system function are known as immunomodulators. These molecules, which include endogenous proteins (e.g., cytokines, cytokine receptors, and intracellular signal transduction molecules), molecules derived from microorganisms, and synthetic agents, may exert their modulatory effects at one or more stages of the immune response, such as antigen recognition, stimulation of cytokine production and release, and/or activation/differentiation of lymphocytes and myeloid cells. Immunomodulators may enhance (immunoprophylaxis, immunostimulation), restore (immunosubstitution, immunorestitution) or suppress (immunosuppression, immunodeviation) immunological functions or activities.

25 Immunomodulatory compounds have many important applications in clinical practice. For example, immunosuppressing agents (which attenuate or prevent unwanted immune responses) can be used to prevent immediate hypersensitivity reactions such as asthma and allergic reactions. A mechanism of action common to many immunosuppressants is the inhibition of T cell activation and/or differentiation. Antilymphocyte antibodies have also been used to attenuate immune system functions. Currently used immunosuppressive agents can produce a number of side effects, which limit their use. Among the most serious secondary effects include kidney and liver toxicity, increased risk of infection, hyperglycemia, neoplasia, and osteoporosis (see, e.g., Freeman, Clin. Biochem. 24(1):9-14 (1991); Mitchison, Dig. Dis.11(2):78-101 (1993)). The discovery of new human allergy and/or asthma related polynucleotides, the polypeptides encoded by them, and antibodies that specifically bind these polypeptides, satisfies a need in the art by

providing new compositions that are useful in the diagnosis, treatment, prevention and/or prognosis of disorders of the immune system, including, but not limited to, allergic reactions and conditions, asthma, and related immediate hypersensitivity disorders.

5

Summary of the Invention

The present invention encompasses human secreted proteins/polypeptides, and isolated nucleic acid molecules encoding said proteins/polypeptides, useful for detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating allergic and/or asthmatic diseases and disorders. Antibodies that bind these polypeptides are also encompassed by the present invention;
10 as are vectors, host cells, and recombinant and synthetic methods for producing said polynucleotides, polypeptides, and/or antibodies. The invention further encompasses screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention also encompasses methods and compositions for inhibiting or enhancing the production and function of the polypeptides of the present invention.

15

Detailed Description

Polynucleotides and Polypeptides of the Invention

Description of Table 1A

20 Table 1A summarizes information concerning certain polynucleotides and polypeptides of the invention. The first column provides the gene number in the application for each clone identifier. The second column provides a unique clone identifier, "Clone ID:", for a cDNA clone related to each contig sequence disclosed in Table 1A. Third column, the cDNA Clones identified in the second column were deposited as indicated in the third column (i.e. by ATCC Deposit No:Z
25 and deposit date). Some of the deposits contain multiple different clones corresponding to the same gene. In the fourth column, "Vector" refers to the type of vector contained in the corresponding cDNA Clone identified in the second column. In the fifth column, the nucleotide sequence identified as "NT SEQ ID NO:X" was assembled from partially homologous ("overlapping") sequences obtained from the corresponding cDNA clone identified in the second
30 column and, in some cases, from additional related cDNA clones. The overlapping sequences were assembled into a single contiguous sequence of high redundancy (usually three to five overlapping sequences at each nucleotide position), resulting in a final sequence identified as SEQ ID NO:X. In the sixth column, "Total NT Seq." refers to the total number of nucleotides in the contig sequence identified as SEQ ID NO:X." The deposited clone may contain all or most of
35 these sequences, reflected by the nucleotide position indicated as "5' NT of Clone Seq." (seventh

column) and the "3' NT of Clone Seq." (eighth column) of SEQ ID NO:X. In the ninth column, the nucleotide position of SEQ ID NO:X of the putative start codon (methionine) is identified as "5' NT of Start Codon." Similarly, in column ten, the nucleotide position of SEQ ID NO:X of the predicted signal sequence is identified as "5' NT of First AA of Signal Pep." In the eleventh column, the translated amino acid sequence, beginning with the methionine, is identified as "AA SEQ ID NO:Y," although other reading frames can also be routinely translated using known molecular biology techniques. The polypeptides produced by these alternative open reading frames are specifically contemplated by the present invention.

In the twelfth and thirteenth columns of Table 1A, the first and last amino acid position of SEQ ID NO:Y of the predicted signal peptide is identified as "First AA of Sig Pep" and "Last AA of Sig Pep." In the fourteenth column, the predicted first amino acid position of SEQ ID NO:Y of the secreted portion is identified as "Predicted First AA of Secreted Portion". The amino acid position of SEQ ID NO:Y of the last amino acid encoded by the open reading frame is identified in the fifteenth column as "Last AA of ORF".

SEQ ID NO:X (where X may be any of the polynucleotide sequences disclosed in the sequence listing) and the translated SEQ ID NO:Y (where Y may be any of the polypeptide sequences disclosed in the sequence listing) are sufficiently accurate and otherwise suitable for a variety of uses well known in the art and described further below. For instance, SEQ ID NO:X is useful for designing nucleic acid hybridization probes that will detect nucleic acid sequences contained in SEQ ID NO:X or the cDNA contained in the deposited clone. These probes will also hybridize to nucleic acid molecules in biological samples, thereby enabling a variety of forensic and diagnostic methods of the invention. Similarly, polypeptides identified from SEQ ID NO:Y may be used, for example, to generate antibodies which bind specifically to proteins containing the polypeptides and the secreted proteins encoded by the cDNA clones identified in Table 1A and/or elsewhere herein

Nevertheless, DNA sequences generated by sequencing reactions can contain sequencing errors. The errors exist as misidentified nucleotides, or as insertions or deletions of nucleotides in the generated DNA sequence. The erroneously inserted or deleted nucleotides cause frame shifts in the reading frames of the predicted amino acid sequence. In these cases, the predicted amino acid sequence diverges from the actual amino acid sequence, even though the generated DNA sequence may be greater than 99.9% identical to the actual DNA sequence (for example, one base insertion or deletion in an open reading frame of over 1000 bases).

Accordingly, for those applications requiring precision in the nucleotide sequence or the amino acid sequence, the present invention provides not only the generated nucleotide sequence identified as SEQ ID NO:X, and the predicted translated amino acid sequence identified as SEQ

ID NO:Y, but also a sample of plasmid DNA containing a human cDNA of the invention deposited with the ATCC, as set forth in Table 1A. The nucleotide sequence of each deposited plasmid can readily be determined by sequencing the deposited plasmid in accordance with known methods

5 The predicted amino acid sequence can then be verified from such deposits. Moreover, the amino acid sequence of the protein encoded by a particular plasmid can also be directly determined by peptide sequencing or by expressing the protein in a suitable host cell containing the deposited human cDNA, collecting the protein, and determining its sequence.

Also provided in Table 1A is the name of the vector which contains the cDNA plasmid.
10 Each vector is routinely used in the art. The following additional information is provided for convenience.

Vectors Lambda Zap (U.S. Patent Nos. 5,128,256 and 5,286,636), Uni-Zap XR (U.S. Patent Nos. 5,128, 256 and 5,286,636), Zap Express (U.S. Patent Nos. 5,128,256 and 5,286,636), pBluescript (pBS) (Short, J. M. et al., *Nucleic Acids Res.* 16:7583-7600 (1988); Altting-Mees, M. A. and Short, J. M., *Nucleic Acids Res.* 17:9494 (1989)) and pBK (Altting-Mees, M. A. et al., *Strategies* 5:58-61 (1992)) are commercially available from Stratagene Cloning Systems, Inc., 11011 N. Torrey Pines Road, La Jolla, CA, 92037. pBS contains an ampicillin resistance gene and pBK contains a neomycin resistance gene. Phagemid pBS may be excised from the Lambda Zap and Uni-Zap XR vectors, and phagemid pBK may be excised from the Zap Express vector. Both
20 phagemids may be transformed into *E. coli* strain XL-1 Blue, also available from Stratagene

Vectors pSport1, pCMVSPORT 1.0, pCMVSPORT 2.0 and pCMVSPORT 3.0, were obtained from Life Technologies, Inc., P. O. Box 6009, Gaithersburg, MD 20897. All Sport vectors contain an ampicillin resistance gene and may be transformed into *E. coli* strain DH10B, also available from Life Technologies. See, for instance, Gruber, C. E., et al., *Focus* 15:59 (1993). Vector lafmid BA (Bento Soares, Columbia University, New York, NY) contains an ampicillin resistance gene and can be transformed into *E. coli* strain XL-1 Blue. Vector pCR[®]2.1, which is available from Invitrogen, 1600 Faraday Avenue, Carlsbad, CA 92008, contains an ampicillin resistance gene and may be transformed into *E. coli* strain DH10B, available from Life Technologies. See, for instance, Clark, J. M., *Nuc. Acids Res.* 16:9677-9686 (1988) and Mead, D. et al., *Bio/Technology* 9: (1991).
30

The present invention also relates to the genes corresponding to SEQ ID NO:X, SEQ ID NO:Y, and/or a deposited cDNA (cDNA Clone ID). The corresponding gene can be isolated in accordance with known methods using the sequence information disclosed herein. Such methods include, but are not limited to, preparing probes or primers from the disclosed sequence and
35 identifying or amplifying the corresponding gene from appropriate sources of genomic material.

Also provided in the present invention are allelic variants, orthologs, and/or species homologs. Procedures known in the art can be used to obtain full-length genes, allelic variants, splice variants, full-length coding portions, orthologs, and/or species homologs of genes corresponding to SEQ ID NO:X and SEQ ID NO:Y using information from the sequences disclosed herein or the clones deposited with the ATCC. For example, allelic variants and/or species homologs may be isolated and identified by making suitable probes or primers from the sequences provided herein and screening a suitable nucleic acid source for allelic variants and/or the desired homologue.

The present invention provides a polynucleotide comprising, or alternatively consisting of, the nucleic acid sequence of SEQ ID NO:X and/or a cDNA contained in ATCC Deposit No.Z. The present invention also provides a polypeptide comprising, or alternatively, consisting of, the polypeptide sequence of SEQ ID NO:Y, a polypeptide encoded by SEQ ID NO:X, and/or a polypeptide encoded by a cDNA contained in ATCC deposit No.Z. Polynucleotides encoding a polypeptide comprising, or alternatively consisting of the polypeptide sequence of SEQ ID NO:Y, a polypeptide encoded by SEQ ID NO:X and/or a polypeptide encoded by the cDNA contained in ATCC Deposit No.Z, are also encompassed by the invention. The present invention further encompasses a polynucleotide comprising, or alternatively consisting of the complement of the nucleic acid sequence of SEQ ID NO:X, and/or the complement of the coding strand of the cDNA contained in ATCC Deposit No.Z.

Description of Table 1B (Comprised of Tables 1B.1 and 1B.2)

Table 1B.1 and Table 1B.2 summarize some of the polynucleotides encompassed by the invention (including cDNA clones related to the sequences (Clone ID:), contig sequences (contig identifier (Contig ID:)) and contig nucleotide sequence identifiers (SEQ ID NO:X)) and further summarizes certain characteristics of these polynucleotides and the polypeptides encoded thereby. The first column of Tables 1B.1 and 1B.2 provide the gene numbers in the application for each clone identifier. The second column of Tables 1B.1 and 1B.2 provide unique clone identifiers, "Clone ID:", for cDNA clones related to each contig sequence disclosed in Table 1A and/or Table 1B. The third column of Tables 1B.1 and 1B.2 provide unique contig identifiers, "Contig ID:" for each of the contig sequences disclosed in these tables. The fourth column of Tables 1B.1 and 1B.2 provide the sequence identifiers, "SEQ ID NO:X", for each of the contig sequences disclosed in Table 1A and/or 1B.

Table 1B.1

The fifth column of Table 1B.1, "ORF (From-To)", provides the location (i.e., nucleotide position numbers) within the polynucleotide sequence of SEQ ID NO:X that delineates

the preferred open reading frame (ORF) that encodes the amino acid sequence shown in the sequence listing and referenced in Table 1B.1 as SEQ ID NO:Y (column 6). Column 7 of Table 1B.1 lists residues comprising predicted epitopes contained in the polypeptides encoded by each of the preferred ORFs (SEQ ID NO:Y). Identification of potential immunogenic regions was performed according to the method of Jameson and Wolf (CABIOS, 4; 181-186 (1988)); specifically, the Genetics Computer Group (GCG) implementation of this algorithm, embodied in the program PEPTIDESTRUCTURE (Wisconsin Package v10.0, Genetics Computer Group (GCG), Madison, Wisc.). This method returns a measure of the probability that a given residue is found on the surface of the protein. Regions where the antigenic index score is greater than 0.9 over at least 6 amino acids are indicated in Table 1B.1 as "Predicted Epitopes". In particular embodiments, polypeptides of the invention comprise, or alternatively consist of, one, two, three, four, five or more of the predicted epitopes described in Table 1B.1. It will be appreciated that depending on the analytical criteria used to predict antigenic determinants, the exact address of the determinant may vary slightly. Column 8 of Table 1B.1 ("Cytologic Band") provides the chromosomal location of polynucleotides corresponding to SEQ ID NO:X. Chromosomal location was determined by finding exact matches to EST and cDNA sequences contained in the NCBI (National Center for Biotechnology Information) UniGene database. Given a presumptive chromosomal location, disease locus association was determined by comparison with the Morbid Map, derived from Online Mendelian Inheritance in Man (Online Mendelian Inheritance in Man, OMIM™. McKusick-Nathans Institute for Genetic Medicine, Johns Hopkins University (Baltimore, MD) and National Center for Biotechnology Information, National Library of Medicine (Bethesda, MD) 2000. World Wide Web URL: <http://www.ncbi.nlm.nih.gov/omim/>). If the putative chromosomal location of the Query overlaps with the chromosomal location of a Morbid Map entry, an OMIM identification number is disclosed in Table 1B.1, column 9 labeled "OMIM Disease Reference(s)". A key to the OMIM reference identification numbers is provided in Table 5.

Table 1B.2

Column 5 of Table 1B.2, "Tissue Distribution" shows the expression profile of tissue, cells, and/or cell line libraries which express the polynucleotides of the invention. The first code number shown in Table 1B.2 column 5 (preceding the colon), represents the tissue/cell source identifier code corresponding to the key provided in Table 4. Expression of these polynucleotides was not observed in the other tissues and/or cell libraries tested. The second number in column 5 (following the colon), represents the number of times a sequence corresponding to the reference polynucleotide sequence (e.g., SEQ ID NO:X) was identified in the corresponding tissue/cell source. Those tissue/cell source identifier codes in which the first two letters are "AR" designate

information generated using DNA array technology. Utilizing this technology, cDNAs were amplified by PCR and then transferred, in duplicate, onto the array. Gene expression was assayed through hybridization of first strand cDNA probes to the DNA array. cDNA probes were generated from total RNA extracted from a variety of different tissues and cell lines. Probe synthesis was performed in the presence of ³³P dCTP, using oligo(dT) to prime reverse transcription. After hybridization, high stringency washing conditions were employed to remove non-specific hybrids from the array. The remaining signal, emanating from each gene target, was measured using a Phosphorimager. Gene expression was reported as Phosphor Stimulating Luminescence (PSL) which reflects the level of phosphor signal generated from the probe hybridized to each of the targets represented on the array. A local background signal subtraction was performed before the total signal generated from each array was used to normalize gene expression between the different hybridizations. The value presented after "[array code]:" represents the mean of the duplicate values, following background subtraction and probe normalization. One of skill in the art could routinely use this information to identify normal and/or diseased tissue(s) which show a predominant expression pattern of the corresponding polynucleotide of the invention or to identify polynucleotides which show predominant and/or specific tissue and/or cell expression.

Description of Table 1C

Table 1C summarizes additional polynucleotides encompassed by the invention (including cDNA clones related to the sequences (Clone ID:), contig sequences (contig identifier (Contig ID:) contig nucleotide sequence identifiers (SEQ ID NO:X)), and genomic sequences (SEQ ID NO:B). The first column provides a unique clone identifier, "Clone ID:", for a cDNA clone related to each contig sequence. The second column provides the sequence identifier, "SEQ ID NO:X", for each contig sequence. The third column provides a unique contig identifier, "Contig ID:" for each contig sequence. The fourth column, provides a BAC identifier "BAC ID NO:A" for the BAC clone referenced in the corresponding row of the table. The fifth column provides the nucleotide sequence identifier, "SEQ ID NO:B" for a fragment of the BAC clone identified in column four of the corresponding row of the table. The sixth column, "Exon From-To", provides the location (i.e., nucleotide position numbers) within the polynucleotide sequence of SEQ ID NO:B which delineate certain polynucleotides of the invention that are also exemplary members of polynucleotide sequences that encode polypeptides of the invention (e.g., polypeptides containing amino acid sequences encoded by the polynucleotide sequences delineated in column six, and fragments and variants thereof).

Description of Table 1D

Table 1D: In preferred embodiments, the present invention encompasses a method of detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating allergic and/or asthmatic diseases and disorders; comprising administering to a patient in which such treatment, prevention, or amelioration is desired a protein, nucleic acid, or antibody of the invention (or fragment or variant thereof) represented by Table 1A, Table 1B, and Table 1C, in an amount effective to detect, prevent, diagnose, prognosticate, treat, and/or ameliorate the disease or disorder.

As indicated in Table 1D, the polynucleotides, polypeptides, agonists, or antagonists of the present invention (including antibodies) can be used in assays to test for one or more biological activities. If these polynucleotides and polypeptides do exhibit activity in a particular assay, it is likely that these molecules may be involved in the diseases associated with the biological activity. Thus, the polynucleotides or polypeptides, or agonists or antagonists thereof (including antibodies) could be used to treat the associated disease.

Table 1D provides information related to biological activities for polynucleotides and polypeptides of the invention (including antibodies, agonists, and/or antagonists thereof). Table 1D also provides information related to assays which may be used to test polynucleotides and polypeptides of the invention (including antibodies, agonists, and/or antagonists thereof) for the corresponding biological activities. The first column ("Gene No.") provides the gene number in the application for each clone identifier. The second column ("cDNA Clone ID:") provides the unique clone identifier for each clone as previously described and indicated in Tables 1A, 1B, and 1C. The third column ("AA SEQ ID NO:Y") indicates the Sequence Listing SEQ ID Number for polypeptide sequences encoded by the corresponding cDNA clones (also as indicated in Tables 1A, 1B, and 2). The fourth column ("Biological Activity") indicates a biological activity corresponding to the indicated polypeptides (or polynucleotides encoding said polypeptides). The fifth column ("Exemplary Activity Assay") further describes the corresponding biological activity and provides information pertaining to the various types of assays that may be performed to test, demonstrate, or quantify the corresponding biological activity. Table 1D describes the use of FMAT technology, *inter alia*, for testing or demonstrating various biological activities. Fluorometric microvolume assay technology (FMAT) is a fluorescence-based system that provides a means to perform nonradioactive cell- and bead-based assays to detect activation of cell signal transduction pathways. This technology was designed specifically for ligand binding and immunological assays. Using this technology, fluorescent cells or beads at the bottom of the well are detected as localized areas of concentrated fluorescence using a data processing system.

Unbound fluorophore comprising the background signal is ignored, allowing for a wide variety of homogeneous assays. FMAT technology may be used for peptide ligand binding assays, immunofluorescence, apoptosis, cytotoxicity, and bead-based immunocapture assays. *See*, Miraglia S et. al., "Homogeneous cell and bead based assays for highthroughput screening using
5 fluorometric microvolume assay technology," *Journal of Biomolecular Screening*; 4:193-204 (1999). In particular, FMAT technology may be used to test, confirm, and/or identify the ability of polypeptides (including polypeptide fragments and variants) to activate signal transduction pathways. For example, FMAT technology may be used to test, confirm, and/or identify the ability of polypeptides to upregulate production of immunomodulatory proteins (such as, for
10 example, interleukins, GM-CSF, Rantes, and Tumor Necrosis factors, as well as other cellular regulators (e.g. insulin)).

Table 1D also describes the use of kinase assays for testing, demonstrating, or quantifying biological activity. In this regard, the phosphorylation and de-phosphorylation of specific amino acid residues (e.g. Tyrosine, Serine, Threonine) on cell-signal transduction proteins provides a fast,
15 reversible means for activation and de-activation of cellular signal transduction pathways. Moreover, cell signal transduction via phosphorylation/de-phosphorylation is crucial to the regulation of a wide variety of cellular processes (e.g. proliferation, differentiation, migration, apoptosis, etc.). Accordingly, kinase assays provide a powerful tool useful for testing, confirming, and/or identifying polypeptides (including polypeptide fragments and variants) that mediate cell
20 signal transduction events via protein phosphorylation. *See e.g.*, Forrer, P., Tamaskovic R., and Jaussi, R. "Enzyme-Linked Immunosorbent Assay for Measurement of JNK, ERK, and p38 Kinase Activities" *Biol. Chem.* 379(8-9): 1101-1110 (1998).

Description of Table 1E

Polynucleotides encoding polypeptides of the present invention can be used in assays to test for one or more biological activities. One such biological activity which may be tested includes the ability of polynucleotides and polypeptides of the invention to stimulate up-regulation or down-regulation of expression of particular genes and proteins. Hence, if polynucleotides and polypeptides of the present invention exhibit activity in altering particular gene and protein
30 expression patterns, it is likely that these polynucleotides and polypeptides of the present invention may be involved in, or capable of effecting changes in, diseases associated with the altered gene and protein expression profiles. Hence, polynucleotides, polypeptides, or antibodies of the present invention could be used to treat said associated diseases.

TaqMan® assays may be performed to assess the ability of polynucleotides (and
35 polypeptides they encode) to alter the expression pattern of particular "target" genes. TaqMan®

reactions are performed to evaluate the ability of a test agent to induce or repress expression of specific genes in different cell types. TaqMan® gene expression quantification assays (“TaqMan® assays”) are well known to, and routinely performed by, those of ordinary skill in the art. TaqMan® assays are performed in a two step reverse transcription / polymerase chain reaction (RT-PCR). In the first (RT) step, cDNA is reverse transcribed from total RNA samples using random hexamer primers. In the second (PCR) step, PCR products are synthesized from the cDNA using gene specific primers.

To quantify gene expression the Taqman® PCR reaction exploits the 5’ nuclease activity of AmpliTaq Gold® DNA Polymerase to cleave a Taqman® probe (distinct from the primers) during PCR. The Taqman® probe contains a reporter dye at the 5’-end of the probe and a quencher dye at the 3’ end of the probe. When the probe is intact, the proximity of the reporter dye to the quencher dye results in suppression of the reporter fluorescence. During PCR, if the target of interest is present, the probe specifically anneals between the forward and reverse primer sites. AmpliTaq Fold DNA Polymerase then cleaves the probe between the reporter and quencher when the probe hybridizes to the target, resulting in increased fluorescence of the reporter (see Figure 2). Accumulation of PCR products is detected directly by monitoring the increase in fluorescence of the reporter dye.

After the probe fragments are displaced from the target, polymerization of the strand continues. The 3’-end of the probe is blocked to prevent extension of the probe during PCR. This process occurs in every cycle and does not interfere with the exponential accumulation of product. The increase in fluorescence signal is detected only if the target sequence is complementary to the probe and is amplified during PCR. Because of these requirements, any nonspecific amplification is not detected.

For test sample preparation, vector controls or constructs containing the coding sequence for the gene of interest are transfected into cells, such as for example 293T cells, and supernatants collected after 48 hours. For cell treatment and RNA isolation, multiple primary human cells or human cell lines are used; such cells may include but are not limited to, Normal Human Dermal Fibroblasts, Aortic Smooth Muscle, Human Umbilical Vein Endothelial Cells, HepG2, Daudi, Jurkat, U937, Caco, and THP-1 cell lines. Cells are plated in growth media and growth is arrested by culturing without media change for 3 days, or by switching cells to low serum media and incubating overnight. Cells are treated for 1, 6, or 24 hours with either vector control supernatant or sample supernatant (or purified/partially purified protein preparations in buffer). Total RNA is isolated; for example, by using Trizol extraction or by using the Ambion RNAqueous(TM)-4PCR RNA isolation system. Expression levels of multiple genes are analyzed using TAQMAN, and expression in the test sample is compared to control vector samples to identify genes induced or

repressed. Each of the above described techniques are well known to, and routinely performed by, those of ordinary skill in the art.

Table 1E indicates particular disease classes and preferred indications for which polynucleotides, polypeptides, or antibodies of the present invention may be used in detecting, diagnosing, preventing, treating and/or ameliorating said diseases and disorders based on "target" gene expression patterns which may be up- or down-regulated by polynucleotides (and the encoded polypeptides) corresponding to each indicated cDNA Clone ID (shown in Table 1E, Column 2).

Thus, in preferred embodiments, the present invention encompasses a method of detecting, diagnosing, preventing, treating, and/or ameliorating a disease or disorder listed in the "Disease Class" and/or "Preferred Indication" columns of Table 1E; comprising administering to a patient in which such detection, diagnosis, prevention, or treatment is desired a protein, nucleic acid, or antibody of the invention (or fragment or variant thereof) in an amount effective to detect, diagnose, prevent, treat, or ameliorate the disease or disorder. The first and second columns of Table 1D show the "Gene No." and "cDNA Clone ID No.", respectively, indicating certain nucleic acids and proteins (or antibodies against the same) of the invention (including polynucleotide, polypeptide, and antibody fragments or variants thereof) that may be used in detecting, diagnosing, preventing, treating, or ameliorating the disease(s) or disorder(s) indicated in column 6 and as indicated in the corresponding row in the "Disease Class" or "Preferred Indication" Columns of Table 1E.

In another embodiment, the present invention also encompasses methods of detecting, diagnosing, preventing, treating, or ameliorating a disease or disorder listed in the "Disease Class" or "Preferred Indication" Columns of Table 1E; comprising administering to a patient combinations of the proteins, nucleic acids, or antibodies of the invention (or fragments or variants thereof), sharing similar indications as shown in the corresponding rows in the "Disease Class" or "Preferred Indication" Columns of Table 1E.

The "Disease Class" Column of Table 1E provides a categorized descriptive heading for diseases, disorders, and/or conditions (more fully described below) that may be detected, diagnosed, prevented, treated, or ameliorated by a protein, nucleic acid, or antibody of the invention (or fragment or variant thereof).

The "Preferred Indication" Column of Table 1E describes diseases, disorders, and/or conditions that may be detected, diagnosed, prevented, treated, or ameliorated by a protein, nucleic acid, or antibody of the invention (or fragment or variant thereof).

The "Cell Line" and "Exemplary Targets" Columns of Table 1E indicate particular cell lines and target genes, respectively, which may show altered gene expression patterns (i.e., up- or

down-regulation of the indicated target gene) in Taqman assays, performed as described above, utilizing polynucleotides of the cDNA Clone ID shown in the corresponding row. Alteration of expression patterns of the indicated "Exemplary Target" genes is correlated with a particular "Disease Class" and/or "Preferred Indication" as shown in the corresponding row under the
5 respective column headings.

The "Exemplary Accessions" Column indicates GenBank Accessions (available online through the National Center for Biotechnology Information (NCBI) at <http://www.ncbi.nlm.nih.gov/>) which correspond to the "Exemplary Targets" shown in the adjacent row.

10 The recitation of "Cancer" in the "Disease Class" Column indicates that the corresponding nucleic acid and protein, or antibody against the same, of the invention (or fragment or variant thereof) may be used for example, to detect, diagnose, prevent, treat, and/or ameliorate neoplastic diseases and/or disorders (e.g., leukemias, cancers, etc., as described below under "Hyperproliferative Disorders").

15 The recitation of "Immune" in the "Disease Class" column indicates that the corresponding nucleic acid and protein, or antibody against the same, of the invention (or fragment or variant thereof), may be used for example, to detect, diagnose, prevent, treat, and/or ameliorate diseases and/or disorders relating to neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as described below under "Immune
20 Activity" "Cardiovascular Disorders" and/or "Blood-Related Disorders"), and infections (e.g., as described below under "Infectious Disease").

The recitation of "Angiogenesis" in the "Disease Class" column indicates that the corresponding nucleic acid and protein, or antibody against the same, of the invention (or fragment or variant thereof), may be used for example, to detect, diagnose, treat, prevent, and/or ameliorate
25 diseases and/or disorders relating to neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), diseases and/or disorders of the cardiovascular system (e.g., as described below under "Cardiovascular Disorders"), diseases and/or disorders involving cellular and genetic abnormalities (e.g., as described below under "Diseases at the Cellular Level"), diseases and/or disorders involving angiogenesis (e.g., as described below under "Anti-
30 Angiogenesis Activity"), to promote or inhibit cell or tissue regeneration (e.g., as described below under "Regeneration"), or to promote wound healing (e.g., as described below under "Wound Healing and Epithelial Cell Proliferation").

The recitation of "Diabetes" in the "Disease Class" column indicates that the corresponding nucleic acid and protein, or antibody against the same, of the invention (or fragment
35 or variant thereof), may be used for example, to detect, diagnose, treat, prevent, and/or ameliorate

diabetes (including diabetes mellitus types I and II), as well as diseases and/or disorders associated with, or consequential to, diabetes (e.g. as described below under "Endocrine Disorders," "Renal Disorders," and "Gastrointestinal Disorders").

5 **Description of Table 2**

Table 2 summarizes homology and features of some of the polypeptides of the invention. The first column provides a unique clone identifier, "Clone ID:", corresponding to a cDNA clone disclosed in Table 1A or Table 1B. The second column provides the unique contig identifier, "Contig ID:" corresponding to contigs in Table 1B and allowing for correlation with the
10 information in Table 1B. The third column provides the sequence identifier, "SEQ ID NO:X", for the contig polynucleotide sequence. The fourth column provides the analysis method by which the homology/identity disclosed in the Table was determined. Comparisons were made between polypeptides encoded by the polynucleotides of the invention and either a non-redundant protein database (herein referred to as "NR"), or a database of protein families (herein referred to as
15 "PFAM") as further described below. The fifth column provides a description of the PFAM/NR hit having a significant match to a polypeptide of the invention. Column six provides the accession number of the PFAM/NR hit disclosed in the fifth column. Column seven, "Score/Percent Identity", provides a quality score or the percent identity, of the hit disclosed in columns five and six. Columns 8 and 9, "NT From" and "NT To" respectively, delineate the
20 polynucleotides in "SEQ ID NO:X" that encode a polypeptide having a significant match to the PFAM/NR database as disclosed in the fifth and sixth columns. In specific embodiments polypeptides of the invention comprise, or alternatively consist of, an amino acid sequence encoded by a polynucleotide in SEQ ID NO:X as delineated in columns 8 and 9, or fragments or variants thereof.

25

Description of Table 3

Table 3 provides polynucleotide sequences that may be disclaimed according to certain embodiments of the invention. The first column provides a unique clone identifier, "Clone ID", for a cDNA clone related to contig sequences disclosed in Table 1B. The second column provides
30 the sequence identifier, "SEQ ID NO:X", for contig sequences disclosed in Table 1A and/or Table 1B. The third column provides the unique contig identifier, "Contig ID:", for contigs disclosed in Table 1B. The fourth column provides a unique integer 'a' where 'a' is any integer between 1 and the final nucleotide minus 15 of SEQ ID NO:X, and the fifth column provides a unique integer 'b' where 'b' is any integer between 15 and the final nucleotide of SEQ ID NO:X, where both a and b
35 correspond to the positions of nucleotide residues shown in SEQ ID NO:X, and where b is greater

than or equal to $a + 14$. For each of the polynucleotides shown as SEQ ID NO:X, the uniquely defined integers can be substituted into the general formula of $a-b$, and used to describe polynucleotides which may be preferably excluded from the invention. In certain embodiments, preferably excluded from the invention are at least one, two, three, four, five, ten, or more of the polynucleotide sequence(s) having the accession number(s) disclosed in the sixth column of this Table (including for example, published sequence in connection with a particular BAC clone). In further embodiments, preferably excluded from the invention are the specific polynucleotide sequence(s) contained in the clones corresponding to at least one, two, three, four, five, ten, or more of the available material having the accession numbers identified in the sixth column of this Table (including for example, the actual sequence contained in an identified BAC clone).

Description of Table 4

Table 4 provides a key to the tissue/cell source identifier code disclosed in Table 1B.2, column 5. Column 1 of Table 4 provides the tissue/cell source identifier code disclosed in Table 1B.2, column 5. Columns 2-5 provide a description of the tissue or cell source. Note that "Description" and "Tissue" sources (i.e. columns 2 and 3) having the prefix "a_" indicates organs, tissues, or cells derived from "adult" sources. Codes corresponding to diseased tissues are indicated in column 6 with the word "disease." The use of the word "disease" in column 6 is non-limiting. The tissue or cell source may be specific (e.g. a neoplasm), or may be disease-associated (e.g., a tissue sample from a normal portion of a diseased organ). Furthermore, tissues and/or cells lacking the "disease" designation may still be derived from sources directly or indirectly involved in a disease state or disorder, and therefore may have a further utility in that disease state or disorder. In numerous cases where the tissue/cell source is a library, column 7 identifies the vector used to generate the library.

Description of Table 5

Table 5 provides a key to the OMIM reference identification numbers disclosed in Table 1B.1, column 9. OMIM reference identification numbers (Column 1) were derived from Online Mendelian Inheritance in Man (Online Mendelian Inheritance in Man, OMIM. McKusick-Nathans Institute for Genetic Medicine, Johns Hopkins University (Baltimore, MD) and National Center for Biotechnology Information, National Library of Medicine, (Bethesda, MD) 2000. World Wide Web URL: <http://www.ncbi.nlm.nih.gov/omim/>). Column 2 provides diseases associated with the cytologic band disclosed in Table 1B.1, column 8, as determined using the Morbid Map database.

Description of Table 6

Table 6 summarizes some of the ATCC Deposits, Deposit dates, and ATCC designation numbers of deposits made with the ATCC in connection with the present application. These deposits were made in addition to those described in the Table 1A.

5 **Description of Table 7**

Table 7 shows the cDNA libraries sequenced, and ATCC designation numbers and vector information relating to these cDNA libraries.

10 The first column shows the first four letters indicating the Library from which each library clone was derived. The second column indicates the catalogued tissue description for the corresponding libraries. The third column indicates the vector containing the corresponding clones. The fourth column shows the ATCC deposit designation for each library clone as indicated by the deposit information in Table 6.

15 **Definitions**

15 The following definitions are provided to facilitate understanding of certain terms used throughout this specification.

20 In the present invention, "isolated" refers to material removed from its original environment (e.g., the natural environment if it is naturally occurring), and thus is altered "by the hand of man" from its natural state. For example, an isolated polynucleotide could be part of a vector or a composition of matter, or could be contained within a cell, and still be "isolated" because that vector, composition of matter, or particular cell is not the original environment of the polynucleotide. The term "isolated" does not refer to genomic or cDNA libraries, whole cell total or mRNA preparations, genomic DNA preparations (including those separated by electrophoresis and transferred onto blots), sheared whole cell genomic DNA preparations or other compositions
25 where the art demonstrates no distinguishing features of the polynucleotide/sequences of the present invention.

30 In the present invention, a "secreted" protein refers to those proteins capable of being directed to the ER, secretory vesicles, or the extracellular space as a result of a signal sequence, as well as those proteins released into the extracellular space without necessarily containing a signal sequence. If the secreted protein is released into the extracellular space, the secreted protein can undergo extracellular processing to produce a "mature" protein. Release into the extracellular space can occur by many mechanisms, including exocytosis and proteolytic cleavage.

35 As used herein, a "polynucleotide" refers to a molecule having a nucleic acid sequence encoding SEQ ID NO:Y or a fragment or variant thereof (e.g., the polypeptide delineated in columns fourteen and fifteen of Table 1A); a nucleic acid sequence contained in SEQ ID NO:X (as

described in column 5 of Table 1A and/or column 3 of Table 1B) or the complement thereof; a cDNA sequence contained in Clone ID: (as described in column 2 of Table 1A and/or Table 1B and contained within a library deposited with the ATCC); a nucleotide sequence encoding the polypeptide encoded by a nucleotide sequence in SEQ ID NO:B as defined in column 6 (EXON From-To) of Table 1C or a fragment or variant thereof; or a nucleotide coding sequence in SEQ ID NO:B as defined in column 6 of Table 1C or the complement thereof. For example, the polynucleotide can contain the nucleotide sequence of the full-length cDNA sequence, including the 5' and 3' untranslated sequences, the coding region, as well as fragments, epitopes, domains, and variants of the nucleic acid sequence. Moreover, as used herein, a "polypeptide" refers to a molecule having an amino acid sequence encoded by a polynucleotide of the invention as broadly defined (obviously excluding poly-Phenylalanine or poly-Lysine peptide sequences which result from translation of a polyA tail of a sequence corresponding to a cDNA).

In the present invention, "SEQ ID NO:X" was often generated by overlapping sequences contained in multiple clones (contig analysis). A representative clone containing all or most of the sequence for SEQ ID NO:X is deposited at Human Genome Sciences, Inc. (HGS) in a catalogued and archived library. As shown, for example, in column 2 of Table 1B, each clone is identified by a cDNA Clone ID (identifier generally referred to herein as Clone ID:). Each Clone ID is unique to an individual clone and the Clone ID is all the information needed to retrieve a given clone from the HGS library. Table 7 provides a list of the deposited cDNA libraries. One can use the Clone ID: to determine the library source by reference to Tables 6 and 7. Table 7 lists the deposited cDNA libraries by name and links each library to an ATCC Deposit. Library names contain four characters, for example, "HTWE." The name of a cDNA clone (Clone ID) isolated from that library begins with the same four characters, for example "HTWEP07". As mentioned below, Table 1A and/or Table 1B correlates the Clone ID names with SEQ ID NO:X. Thus, starting with an SEQ ID NO:X, one can use Tables 1A, 1B, 6, 7, and 9 to determine the corresponding Clone ID, which library it came from and which ATCC deposit the library is contained in. Furthermore, it is possible to retrieve a given cDNA clone from the source library by techniques known in the art and described elsewhere herein. The ATCC is located at 10801 University Boulevard, Manassas, Virginia 20110-2209, USA. The ATCC deposits were made pursuant to the terms of the Budapest Treaty on the international recognition of the deposit of microorganisms for the purposes of patent procedure.

In specific embodiments, the polynucleotides of the invention are at least 15, at least 30, at least 50, at least 100, at least 125, at least 500, or at least 1000 continuous nucleotides but are less than or equal to 300 kb, 200 kb, 100 kb, 50 kb, 15 kb, 10 kb, 7.5kb, 5 kb, 2.5 kb, 2.0 kb, or 1 kb, in length. In a further embodiment, polynucleotides of the invention comprise a portion of the coding

sequences, as disclosed herein, but do not comprise all or a portion of any intron. In another embodiment, the polynucleotides comprising coding sequences do not contain coding sequences of a genomic flanking gene (i.e., 5' or 3' to the gene of interest in the genome). In other embodiments, the polynucleotides of the invention do not contain the coding sequence of more than 1000, 500, 250, 100, 50, 25, 20, 15, 10, 5, 4, 3, 2, or 1 genomic flanking gene(s).

A "polynucleotide" of the present invention also includes those polynucleotides capable of hybridizing, under stringent hybridization conditions, to sequences contained in SEQ ID NO:X, or the complement thereof (e.g., the complement of any one, two, three, four, or more of the polynucleotide fragments described herein), the polynucleotide sequence delineated in columns 7 and 8 of Table 1A or the complement thereof, the polynucleotide sequence delineated in columns 8 and 9 of Table 2 or the complement thereof, and/or cDNA sequences contained in Clone ID: (e.g., the complement of any one, two, three, four, or more of the polynucleotide fragments, or the cDNA clone within the pool of cDNA clones deposited with the ATCC, described herein), and/or the polynucleotide sequence delineated in column 6 of Table 1C or the complement thereof. "Stringent hybridization conditions" refers to an overnight incubation at 42 degree C in a solution comprising 50% formamide, 5x SSC (750 mM NaCl, 75 mM trisodium citrate), 50 mM sodium phosphate (pH 7.6), 5x Denhardt's solution, 10% dextran sulfate, and 20 µg/ml denatured, sheared salmon sperm DNA, followed by washing the filters in 0.1x SSC at about 65 degree C.

Also contemplated are nucleic acid molecules that hybridize to the polynucleotides of the present invention at lower stringency hybridization conditions. Changes in the stringency of hybridization and signal detection are primarily accomplished through the manipulation of formamide concentration (lower percentages of formamide result in lowered stringency); salt conditions, or temperature. For example, lower stringency conditions include an overnight incubation at 37 degree C in a solution comprising 6X SSPE (20X SSPE = 3M NaCl; 0.2M NaH₂PO₄; 0.02M EDTA, pH 7.4), 0.5% SDS, 30% formamide, 100 ug/ml salmon sperm blocking DNA; followed by washes at 50 degree C with 1XSSPE, 0.1% SDS. In addition, to achieve even lower stringency, washes performed following stringent hybridization can be done at higher salt concentrations (e.g. 5X SSC).

Note that variations in the above conditions may be accomplished through the inclusion and/or substitution of alternate blocking reagents used to suppress background in hybridization experiments. Typical blocking reagents include Denhardt's reagent, BLOTTO, heparin, denatured salmon sperm DNA, and commercially available proprietary formulations. The inclusion of specific blocking reagents may require modification of the hybridization conditions described above, due to problems with compatibility.

Of course, a polynucleotide which hybridizes only to polyA⁺ sequences (such as any 3'

terminal polyA⁺ tract of a cDNA shown in the sequence listing), or to a complementary stretch of T (or U) residues, would not be included in the definition of "polynucleotide," since such a polynucleotide would hybridize to any nucleic acid molecule containing a poly (A) stretch or the complement thereof (e.g., practically any double-stranded cDNA clone generated using oligo dT as a primer).

The polynucleotide of the present invention can be composed of any polyribonucleotide or polydeoxribonucleotide, which may be unmodified RNA or DNA or modified RNA or DNA. For example, polynucleotides can be composed of single- and double-stranded DNA, DNA that is a mixture of single- and double-stranded regions, single- and double-stranded RNA, and RNA that is a mixture of single- and double-stranded regions, hybrid molecules comprising DNA and RNA that may be single-stranded or, more typically, double-stranded or a mixture of single- and double-stranded regions. In addition, the polynucleotide can be composed of triple-stranded regions comprising RNA or DNA or both RNA and DNA. A polynucleotide may also contain one or more modified bases or DNA or RNA backbones modified for stability or for other reasons. "Modified" bases include, for example, tritylated bases and unusual bases such as inosine. A variety of modifications can be made to DNA and RNA; thus, "polynucleotide" embraces chemically, enzymatically, or metabolically modified forms.

In specific embodiments, the polynucleotides of the invention are at least 15, at least 30, at least 50, at least 100, at least 125, at least 500, or at least 1000 continuous nucleotides but are less than or equal to 300 kb, 200 kb, 100 kb, 50 kb, 15 kb, 10 kb, 7.5kb, 5 kb, 2.5 kb, 2.0 kb, or 1 kb, in length. In a further embodiment, polynucleotides of the invention comprise a portion of the coding sequences, as disclosed herein, but do not comprise all or a portion of any intron. In another embodiment, the polynucleotides comprising coding sequences do not contain coding sequences of a genomic flanking gene (i.e., 5' or 3' to the gene of interest in the genome). In other embodiments, the polynucleotides of the invention do not contain the coding sequence of more than 1000, 500, 250, 100, 50, 25, 20, 15, 10, 5, 4, 3, 2, or 1 genomic flanking gene(s).

"SEQ ID NO:X" refers to a polynucleotide sequence described in column 5 of Table 1A, while "SEQ ID NO:Y" refers to a polypeptide sequence described in column 10 of Table 1A. SEQ ID NO:X is identified by an integer specified in column 6 of Table 1A. The polypeptide sequence SEQ ID NO:Y is a translated open reading frame (ORF) encoded by polynucleotide SEQ ID NO:X. The polynucleotide sequences are shown in the sequence listing immediately followed by all of the polypeptide sequences. Thus, a polypeptide sequence corresponding to polynucleotide sequence SEQ ID NO:2 is the first polypeptide sequence shown in the sequence listing. The second polypeptide sequence corresponds to the polynucleotide sequence shown as SEQ ID NO:3, and so on.

The polypeptide of the present invention can be composed of amino acids joined to each other by peptide bonds or modified peptide bonds, i.e., peptide isosteres, and may contain amino acids other than the 20 gene-encoded amino acids. The polypeptides may be modified by either natural processes, such as posttranslational processing, or by chemical modification techniques which are well known in the art. Such modifications are well described in basic texts and in more detailed monographs, as well as in a voluminous research literature. Modifications can occur anywhere in a polypeptide, including the peptide backbone, the amino acid side-chains and the amino or carboxyl termini. It will be appreciated that the same type of modification may be present in the same or varying degrees at several sites in a given polypeptide. Also, a given polypeptide may contain many types of modifications. Polypeptides may be branched, for example, as a result of ubiquitination, and they may be cyclic, with or without branching. Cyclic, branched, and branched cyclic polypeptides may result from posttranslation natural processes or may be made by synthetic methods. Modifications include acetylation, acylation, ADP-ribosylation, amidation, covalent attachment of flavin, covalent attachment of a heme moiety, covalent attachment of a nucleotide or nucleotide derivative, covalent attachment of a lipid or lipid derivative, covalent attachment of phosphatidylinositol, cross-linking, cyclization, disulfide bond formation, demethylation, formation of covalent cross-links, formation of cysteine, formation of pyroglutamate, formylation, gamma-carboxylation, glycosylation, GPI anchor formation, hydroxylation, iodination, methylation, myristoylation, oxidation, pegylation, proteolytic processing, phosphorylation, prenylation, racemization, selenoylation, sulfation, transfer-RNA mediated addition of amino acids to proteins such as arginylation, and ubiquitination. (See, for instance, PROTEINS - STRUCTURE AND MOLECULAR PROPERTIES, 2nd Ed., T. E. Creighton, W. H. Freeman and Company, New York (1993); POSTTRANSLATIONAL COVALENT MODIFICATION OF PROTEINS, B. C. Johnson, Ed., Academic Press, New York, pgs. 1-12 (1983); Seifter et al., Meth. Enzymol. 182:626-646 (1990); Rattan et al., Ann. N.Y. Acad. Sci. 663:48-62 (1992)).

"SEQ ID NO:X" refers to a polynucleotide sequence described, for example, in Tables 1A, Table 1B, or Table 2, while "SEQ ID NO:Y" refers to a polypeptide sequence described in column 11 of Table 1A and or Table 1B. SEQ ID NO:X is identified by an integer specified in column 4 of Table 1B. The polypeptide sequence SEQ ID NO:Y is a translated open reading frame (ORF) encoded by polynucleotide SEQ ID NO:X. "Clone ID:" refers to a cDNA clone described in column 2 of Table 1A and/or 1B.

"A polypeptide having functional activity" refers to a polypeptide capable of displaying one or more known functional activities associated with a full-length (complete) protein. Such functional activities include, but are not limited to, biological activity (e.g. activity useful in

treating, preventing and/or ameliorating allergic and/or asthmatic diseases and disorders), antigenicity (ability to bind [or compete with a polypeptide for binding] to an anti-polypeptide antibody), immunogenicity (ability to generate antibody which binds to a specific polypeptide of the invention), ability to form multimers with polypeptides of the invention, and ability to bind to a receptor or ligand for a polypeptide.

The polypeptides of the invention can be assayed for functional activity (e.g. biological activity) using or routinely modifying assays known in the art, as well as assays described herein. Specifically, one of skill in the art may routinely assay secreted polypeptides (including fragments and variants) of the invention for activity using assays as described in the examples section below.

"A polypeptide having biological activity" refers to a polypeptide exhibiting activity similar to, but not necessarily identical to, an activity of a polypeptide of the present invention, including mature forms, as measured in a particular biological assay, with or without dose dependency. In the case where dose dependency does exist, it need not be identical to that of the polypeptide, but rather substantially similar to the dose-dependence in a given activity as compared to the polypeptide of the present invention (i.e., the candidate polypeptide will exhibit greater activity or not more than about 25-fold less and, preferably, not more than about tenfold less activity, and most preferably, not more than about three-fold less activity relative to the polypeptide of the present invention).

TABLES

Table 1A

Table 1A summarizes information concerning certain polynucleotides and polypeptides of the invention. The first column provides the gene number in the application for each clone identifier. The second column provides a unique clone identifier, "Clone ID:", for a cDNA clone related to each contig sequence disclosed in Table 1A. Third column, the cDNA Clones identified in the second column were deposited as indicated in the third column (i.e. by ATCC Deposit No:Z and deposit date). Some of the deposits contain multiple different clones corresponding to the same gene. In the fourth column, "Vector" refers to the type of vector contained in the corresponding cDNA Clone identified in the second column. In the fifth column, the nucleotide sequence identified as "NT SEQ ID NO:X" was assembled from partially homologous ("overlapping") sequences obtained from the corresponding cDNA clone identified in the second column and, in some cases, from additional related cDNA clones. The overlapping sequences were assembled into a single contiguous sequence of high redundancy (usually three to five overlapping sequences at each nucleotide position), resulting in a final sequence identified as SEQ ID NO:X. In the sixth column, "Total NT Seq." refers to the total number of nucleotides in the

contig sequence identified as SEQ ID NO:X.” The deposited clone may contain all or most of these sequences, reflected by the nucleotide position indicated as “5’ NT of Clone Seq.” (seventh column) and the “3’ NT of Clone Seq.” (eighth column) of SEQ ID NO:X. In the ninth column, the nucleotide position of SEQ ID NO:X of the putative start codon (methionine) is identified as “5’ NT of Start Codon.” Similarly, in column ten, the nucleotide position of SEQ ID NO:X of the predicted signal sequence is identified as “5’ NT of First AA of Signal Pep.” In the eleventh column, the translated amino acid sequence, beginning with the methionine, is identified as “AA SEQ ID NO:Y,” although other reading frames can also be routinely translated using known molecular biology techniques. The polypeptides produced by these alternative open reading frames are specifically contemplated by the present invention.

In the twelfth and thirteenth columns of Table 1A, the first and last amino acid position of SEQ ID NO:Y of the predicted signal peptide is identified as “First AA of Sig Pep” and “Last AA of Sig Pep.” In the fourteenth column, the predicted first amino acid position of SEQ ID NO:Y of the secreted portion is identified as “Predicted First AA of Secreted Portion”. The amino acid position of SEQ ID NO:Y of the last amino acid encoded by the open reading frame is identified in the fifteenth column as “Last AA of ORF”.

SEQ ID NO:X (where X may be any of the polynucleotide sequences disclosed in the sequence listing) and the translated SEQ ID NO:Y (where Y may be any of the polypeptide sequences disclosed in the sequence listing) are sufficiently accurate and otherwise suitable for a variety of uses well known in the art and described further below. For instance, SEQ ID NO:X is useful for designing nucleic acid hybridization probes that will detect nucleic acid sequences contained in SEQ ID NO:X or the cDNA contained in the deposited clone. These probes will also hybridize to nucleic acid molecules in biological samples, thereby enabling a variety of forensic and diagnostic methods of the invention. Similarly, polypeptides identified from SEQ ID NO:Y may be used, for example, to generate antibodies which bind specifically to proteins containing the polypeptides and the secreted proteins encoded by the cDNA clones identified in Table 1A and/or elsewhere herein

Nevertheless, DNA sequences generated by sequencing reactions can contain sequencing errors. The errors exist as misidentified nucleotides, or as insertions or deletions of nucleotides in the generated DNA sequence. The erroneously inserted or deleted nucleotides cause frame shifts in the reading frames of the predicted amino acid sequence. In these cases, the predicted amino acid sequence diverges from the actual amino acid sequence, even though the generated DNA sequence may be greater than 99.9% identical to the actual DNA sequence (for example, one base insertion or deletion in an open reading frame of over 1000 bases).

Accordingly, for those applications requiring precision in the nucleotide sequence or the

amino acid sequence, the present invention provides not only the generated nucleotide sequence identified as SEQ ID NO:X, and the predicted translated amino acid sequence identified as SEQ ID NO:Y, but also a sample of plasmid DNA containing a human cDNA of the invention deposited with the ATCC, as set forth in Table 1A. The nucleotide sequence of each deposited
5 plasmid can readily be determined by sequencing the deposited plasmid in accordance with known methods

The predicted amino acid sequence can then be verified from such deposits. Moreover, the amino acid sequence of the protein encoded by a particular plasmid can also be directly determined by peptide sequencing or by expressing the protein in a suitable host cell containing
10 the deposited human cDNA, collecting the protein, and determining its sequence.

Also provided in Table 1A is the name of the vector which contains the cDNA plasmid. Each vector is routinely used in the art. The following additional information is provided for convenience.

Vectors Lambda Zap (U.S. Patent Nos. 5,128,256 and 5,286,636), Uni-Zap XR (U.S.
15 Patent Nos. 5,128, 256 and 5,286,636), Zap Express (U.S. Patent Nos. 5,128,256 and 5,286,636), pBluescript (pBS) (Short, J. M. et al., *Nucleic Acids Res.* 16:7583-7600 (1988); Alting-Mees, M. A. and Short, J. M., *Nucleic Acids Res.* 17:9494 (1989)) and pBK (Alting-Mees, M. A. et al., *Strategies* 5:58-61 (1992)) are commercially available from Stratagene Cloning Systems, Inc., 11011 N. Torrey Pines Road, La Jolla, CA, 92037. pBS contains an ampicillin resistance gene and
20 pBK contains a neomycin resistance gene. Phagemid pBS may be excised from the Lambda Zap and Uni-Zap XR vectors, and phagemid pBK may be excised from the Zap Express vector. Both phagemids may be transformed into *E. coli* strain XL-1 Blue, also available from Stratagene

Vectors pSport1, pCMVSPORT 1.0, pCMVSPORT 2.0 and pCMVSPORT 3.0, were obtained from Life Technologies, Inc., P. O. Box 6009, Gaithersburg, MD 20897. All Sport vectors contain
25 an ampicillin resistance gene and may be transformed into *E. coli* strain DH10B, also available from Life Technologies. See, for instance, Gruber, C. E., et al., *Focus* 15:59 (1993). Vector lafmid BA (Bento Soares, Columbia University, New York, NY) contains an ampicillin resistance gene and can be transformed into *E. coli* strain XL-1 Blue. Vector pCR[®]2.1, which is available from Invitrogen, 1600 Faraday Avenue, Carlsbad, CA 92008, contains an ampicillin resistance
30 gene and may be transformed into *E. coli* strain DH10B, available from Life Technologies. See, for instance, Clark, J. M., *Nuc. Acids Res.* 16:9677-9686 (1988) and Mead, D. et al., *Bio/Technology* 9: (1991).

The present invention also relates to the genes corresponding to SEQ ID NO:X, SEQ ID NO:Y, and/or a deposited cDNA (cDNA Clone ID). The corresponding gene can be isolated in
35 accordance with known methods using the sequence information disclosed herein. Such methods

include, but are not limited to, preparing probes or primers from the disclosed sequence and identifying or amplifying the corresponding gene from appropriate sources of genomic material.

Also provided in the present invention are allelic variants, orthologs, and/or species homologs. Procedures known in the art can be used to obtain full-length genes, allelic variants, splice variants, full-length coding portions, orthologs, and/or species homologs of genes corresponding to SEQ ID NO:X and SEQ ID NO:Y using information from the sequences disclosed herein or the clones deposited with the ATCC. For example, allelic variants and/or species homologs may be isolated and identified by making suitable probes or primers from the sequences provided herein and screening a suitable nucleic acid source for allelic variants and/or the desired homologue.

The present invention provides a polynucleotide comprising, or alternatively consisting of, the nucleic acid sequence of SEQ ID NO:X and/or a cDNA contained in ATCC Deposit No.Z. The present invention also provides a polypeptide comprising, or alternatively, consisting of, the polypeptide sequence of SEQ ID NO:Y, a polypeptide encoded by SEQ ID NO:X, and/or a polypeptide encoded by a cDNA contained in ATCC deposit No.Z. Polynucleotides encoding a polypeptide comprising, or alternatively consisting of the polypeptide sequence of SEQ ID NO:Y, a polypeptide encoded by SEQ ID NO:X and/or a polypeptide encoded by the cDNA contained in ATCC Deposit No.Z, are also encompassed by the invention. The present invention further encompasses a polynucleotide comprising, or alternatively consisting of the complement of the nucleic acid sequence of SEQ ID NO:X, and/or the complement of the coding strand of the cDNA contained in ATCC Deposit No.Z.

TABLE 1A

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
1	HACBT91	203917 04/08/99	Uni-ZAP XR	11	841	1	841		329	116	1	7	8	59
2	HADDE71	203917 04/08/99	pSport1	12	667	1	667	250	250	117	1	28	29	139
3	HADDJ13	203917 04/08/99	pSport1	13	2318	1	2318	347	347	118	1	20	21	30
4	HADMA77	203917 04/08/99	pBluescript	14	1913	763	1913		992	119	1	14	15	23
5	HADMB15	203979 04/29/99	pBluescript	15	330	1	330		238	120	1	11	12	20
6	HAGBQ12	203917 04/08/99	Uni-ZAP XR	16	743	1	743	171	171	121	1	19	20	21
7	HAGCC87	203917 04/08/99	Uni-ZAP XR	17	1592	479	1592	509	509	122	1			9
8	HAGHN57	203917 04/08/99	Uni-ZAP XR	18	2440	843	2440	900	900	123	1			10
9	HAGHR18	203917 04/08/99	Uni-ZAP XR	19	1142	1	1142	28	28	124	1	17	18	32
10	HAQA192	203917 04/08/99	Uni-ZAP XR	20	607	1	602	250	250	125	1	15	16	23
11	HAQBG57	203917 04/08/99	Uni-ZAP XR	21	1048	1	1031		170	126	1	15	16	56

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
12	HAQCE11	203917 04/08/99	Uni-ZAP XR	22	596	1	596		262	127	1			3
13	HBAGD86	203917 04/08/99	pSport1	23	1713	293	1596	521	521	128	1	18	19	19
14	HBGBC29	203917 04/08/99	Uni-ZAP XR	24	1856	764	1829		1016	129	1			2
15	HBJAB02	203917 04/08/99	Uni-ZAP XR	25	1693	1	1665	84	84	130	1	27	28	34
16	HBMUH74	PTA-181 06/07/99	Uni-ZAP XR	26	726	1	726	344	344	131	1	13	14	28
17	HBNAX40	203917 04/08/99	Uni-ZAP XR	27	2793	2455	2793	2497	2497	132	1	18	19	49
18	HBXCX15	203917 04/08/99	ZAP Express	28	1219	1	1219		1148	133	1			1
19	HCDBO32	203917 04/08/99	Uni-ZAP XR	29	2630	1480	2630	1669	1669	134	1	25	26	71
20	HCEEE79	203917 04/08/99	Uni-ZAP XR	30	1052	1	1052	131	131	135	1	15	16	55
21	HCEFZ82	203917 04/08/99	Uni-ZAP XR	31	1811	44	1781	215	215	136	1	16	17	265
22	HCUCF89	203917 04/08/99	ZAP Express	32	530	1	530	189	189	137	1	18	19	29
23	HCWAE64	203917 04/08/99	ZAP Express	33	471	1	471		410	138	1			5
24	HCWUL09	203917 04/08/99	ZAP Express	34	761	3	761	333	333	139	1			11

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
25	HDPDI72	PTA-794 09/27/99	pCMV/Sport 3.0	35	1550	1	1550	23	23	140	1	17	18	120
26	HDPFY18	203918 04/08/99	pCMV/Sport 3.0	36	2187	1	2187	161	161	141	1			7
27	HDPIE44	PTA-794 09/27/99	pCMV/Sport 3.0	37	4115	1	4115	169	169	142	1	35	36	60
28	HDPIU94	203960 04/26/99	pCMV/Sport 3.0	38	2196	21	2196	208	208	143	1	21	22	23
29	HDPPD93	203960 04/26/99	pCMV/Sport 3.0	39	701	1	701	28	28	144	1			12
30	HDTLM18	203960 04/26/99	pCMV/Sport 2.0	40	525	1	525	345	345	145	1	18	19	60
31	HE6CS65	203960 04/26/99	Uni-ZAP XR	41	1526	1	1526		295	146	1	10	11	62
32	HE8BQ49	203960 04/26/99	Uni-ZAP XR	42	1875	12	1875	133	133	147	1			11
33	HE9CY05	203960 04/26/99	Uni-ZAP XR	43	1047	47	1047	55	55	148	1	21	22	235
34	HEAAW94	203979 04/29/99	Uni-ZAP XR	44	924	1	924	189	189	149	1			11
35	HEBFR46	203979 04/29/99	Uni-ZAP XR	45	1304	1	1304	200	200	150	1	26	27	29
36	HEOMC46	PTA-181 06/07/99	pSport1	46	939	1	939		154	151	1	40	41	51
37	HFCDW95	203979 04/29/99	Uni-ZAP XR	47	871	1	871		151	152	1			2

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
38	HFEB017	PTA-181 06/07/99	Uni-ZAP XR	48	990	1	990	136	136	153	1	17	18	27
39	HFUJA29	203960 04/26/99	pSportl	49	1275	110	1275	175	175	154	1	27	28	82
40	HFKFX64	203960 04/26/99	Uni-ZAP XR	50	779	1	779	127	127	155	1			14
41	HGBER72	203960 04/26/99	Uni-ZAP XR	51	1316	1	1316	43	43	156	1	16	17	19
42	HGBGN34	203960 04/26/99	Uni-ZAP XR	52	528	1	528		280	157	1	32	33	48
43	HGLBG15	203960 04/26/99	Uni-ZAP XR	53	778	1	778		191	158	1			26
44	HHFEC39	203960 04/26/99	Uni-ZAP XR	54	1302	1	1302		1211	159	1			1
45	HHSDI53	PTA-181 06/07/99	Uni-ZAP XR	55	1277	1	1277	221	221	160	1	14	15	24
46	HISBA38	203957 04/26/99	pSportl	56	1058	1	1058	169	169	161	1	32	33	36
47	HJPBK28	203957 04/26/99	Uni-ZAP XR	57	989	1	989		256	162	1	21	22	43
48	HKABU43	203959 04/26/99	pCMVSPORT 2.0	58	1919	581	1919	755	755	163	1	20	21	281
49	HLYGE16	203957 04/26/99	pSportl	59	752	1	752	406	406	164	1	17	18	73
50	HLYGY91	203957 04/26/99	pSportl	60	640	1	640	211	211	165	1	20	21	42

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
51	HMCFH60	203957 04/26/99	Uni-ZAP XR	61	443	1	443	211	211	166	1	17	18	48
52	HMIK10	203957 04/26/99	Uni-ZAP XR	62	1064	1	1064	195	195	167	1	22	23	31
53	HMIBD93	203957 04/26/99	Uni-ZAP XR	63	1323	734	1323		983	168	1	27	28	65
54	HMWJF53	203957 04/26/99	Uni-ZAP XR	64	2288	927	2101	1015	1015	169	1	30	31	38
55	HNECL22	203957 04/26/99	Uni-ZAP XR	65	2710	225	2710	472	472	170	1	23	24	34
56	HNFAC50	203957 04/26/99	Uni-ZAP XR	66	1442	428	1442	676	676	171	1	22	23	32
57	HNGEA34	203957 04/26/99	Uni-ZAP XR	67	1103	1	1103		58	172	1	24	25	44
58	HNGIV64	203957 04/26/99	Uni-ZAP XR	68	1047	1	1047		221	173	1			8
59	HNGKT41	203959 04/26/99	Uni-ZAP XR	69	1048	1	1048	415	415	174	1	17	18	45
60	HNGNO53	203959 04/26/99	Uni-ZAP XR	70	825	1	825	467	467	175	1	15	16	34
61	HNHCT47	203959 04/26/99	Uni-ZAP XR	71	621	12	621	73	73	176	1	20	21	39
62	HNHKT74	203959 04/26/99	Uni-ZAP XR	72	817	1	817	127	127	177	1			10
63	HORBS82	203959 04/26/99	Uni-ZAP XR	73	1125	1	1125		21	178	1	19	20	39

Gene No.	cDNA Clone ID	ATCC Deposit No.:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
64	HOUDE92	203918 04/08/99	Uni-ZAP XR	74	1284	1	1282		70	179	1	6	7	88
65	HOUFS04	203959 04/26/99	Uni-ZAP XR	75	2927	457	2882	520	520	180	1	42	43	72
66	HOUHI25	PTA-793 09/27/99	Uni-ZAP XR	76	1249	45	1102	188	188	181	1			20
67	HPCAL26	203917 04/08/99	Uni-ZAP XR	77	3097	803	3097	1021	1021	182	1	23	24	30
68	HPEBA84	203959 04/26/99	Uni-ZAP XR	78	1160	250	1160	533	533	183	1	21	22	36
69	HSAVA08	203918 04/08/99	Uni-ZAP XR	79	1061	1	1061		66	184	1	17	18	26
70	HSAX04	203959 04/26/99	Uni-ZAP XR	80	1287	494	1285		42	185	1	6	7	57
71	HSKDR27	203918 04/08/99	Uni-ZAP XR	81	762	1	762		473	186	1	11	12	27
72	HSQBF66	203918 04/08/99	Uni-ZAP XR	82	1024	1	1024		229	187	1	28	29	66
73	HSRFD18	203959 04/26/99	Uni-ZAP XR	83	1889	1	1793	67	67	188	1	20	21	28
74	HSWBE76	203959 04/26/99	pCMVSPORT 3.0	84	874	250	710	380	380	189	1	34	35	59
75	HT3BF49	203959 04/26/99	Uni-ZAP XR	85	2174	1	2174		306	190	1			4
76	HTEEW69	203959 04/26/99	Uni-ZAP XR	86	1282	110	1263	182	182	191	1	30	31	323

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
77	HTEHU59	203959 04/26/99	Uni-ZAP XR	87	1523	1	1504	170	170	192	1	19	20	34
78	HTEMQ17	203959 04/26/99	Uni-ZAP XR	88	1768	1	1768	446	446	193	1			12
79	HTGBK95	203959 04/26/99	Uni-ZAP XR	89	1131	1	1131	271	271	194	1	12	13	16
80	HTLEM16	203959 04/26/99	Uni-ZAP XR	90	1915	1158	1755	1220	1220	195	1	27	28	69
81	HTNBK13	203959 04/26/99	pBluescript SK-	91	1160	295	1148	534	534	196	1	16	17	21
82	HTODN35	203918 04/08/99	Uni-ZAP XR	92	1594	1	1594	67	67	197	1			14
83	HTPDU17	203959 04/26/99	Uni-ZAP XR	93	2078	1	2078		52	198	1	17	18	33
84	HTTDN24	203959 04/26/99	Uni-ZAP XR	94	1992	856	1992		1024	199	1	13	14	234
85	HTTEE41	203959 04/26/99	Uni-ZAP XR	95	1973	864	1968		1171	200	1			8
86	HTXJD85	203959 04/26/99	Uni-ZAP XR	96	1117	1	1117	211	211	201	1	16	17	31
87	HUVDJ48	203918 04/08/99	Uni-ZAP XR	97	1827	1	1827	196	196	202	1			5
88	HWBBU75	203979 04/29/99	pCMVSPORT 3.0	98	2731	623	2731	783	783	203	1	22	23	51
89	HWHPB78	203959 04/26/99	pCMVSPORT 3.0	99	1346	1	1346	200	200	204	1	23	24	66

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
90	HWLBO67	203959 04/26/99	pSport1	100	536	1	536	42	42	205	1	28	29	39
91	HWLGP26	203959 04/26/99	pSport1	101	1898	1007	1835	1091	1091	206	1	23	24	71
92	HILCA24	203960 04/26/99	pBluescript SK-	102	1982	153	1982	191	191	207	1	29	30	327
92	HILCA24	203960 04/26/99	pBluescript SK-	108	1980	151	1976	189	189	213	1	29	30	327
93	HE2CA60	203960 04/26/99	Uni-ZAP XR	103	3034	1679	3034	1731	1731	208	1			7
93	HE2CA60	203960 04/26/99	Uni-ZAP XR	109	1663	308	1663	360	360	214	1			7
94	HPWTF23	203979 04/29/99	Uni-ZAP XR	104	2008	94	1994	283	283	209	1	29	30	130
94	HPWTF23	203979 04/29/99	Uni-ZAP XR	110	2008	94	1994	283	283	215	1	29	30	130
95	HGCAC19	203960 04/26/99	pSport1	105	5061	23	1475		317	210	1			9
95	HGCAC19	203960 04/26/99	pSport1	111	1771	21	1473		315	216	1			9
95	HGCAC19	203960 04/26/99	pSport1	112	1534	23	1534		317	217	1			9
96	HEQBJ01	203960 04/26/99	pCMVSPORT 3.0	106	2791	2346	2731	2603	2603	211	1			19
96	HEQBJ01	203960 04/26/99	pCMVSPORT 3.0	113	2791	2346	2731	2603	2603	218	1			19

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
96	HEQBJ01	203960 04/26/99	pCMVSPORT 3.0	114	669	1	662	505	505	219	1			19
97	HBJHT01	203917 04/08/99	Uni-ZAP XR	107	1251	1	1251	200	200	212	1	20	21	21
97	HBJHT01	203917 04/08/99	Uni-ZAP XR	115	1252	1	1252	193	193	220	1	21	22	47
98	HAGDW20	203917 04/08/99	Uni-ZAP XR	108	1284	1	1284	238	238	215	1	16	17	17
99	HTLIF11	203959 04/26/99	Uni-ZAP XR	109	1968	860	1968	933	933	216	1	33	34	38

Table 1B (Comprised of Tables 1B.1 and 1B.2)

The first column in Table 1B.1 and Table 1B.2 provides the gene number in the application corresponding to the clone identifier. The second column in Table 1B.1 and Table 1B.2 provides a unique "Clone ID:" for the cDNA clone related to each contig sequence disclosed in Table 1B.1 and Table 1B.2. This clone ID references the cDNA clone which contains at least the 5' most sequence of the assembled contig and at least a portion of SEQ ID NO:X as determined by directly sequencing the referenced clone. The referenced clone may have more sequence than described in the sequence listing or the clone may have less. In the vast majority of cases, however, the clone is believed to encode a full-length polypeptide. In the case where a clone is not full-length, a full-length cDNA can be obtained by methods described elsewhere herein. The third column in Table 1B.1 and Table 1B.2 provides a unique "Contig ID" identification for each contig sequence. The fourth column in Table 1B.1 and Table 1B.2 provides the "SEQ ID NO:" identifier for each of the contig polynucleotide sequences disclosed in Table 1B.

Table 1B.1

The fifth column in Table 1B.1, "ORF (From-To)", provides the location (i.e., nucleotide position numbers) within the polynucleotide sequence "SEQ ID NO:X" that delineate the preferred open reading frame (ORF) shown in the sequence listing and referenced in Table 1B.1, column 6, as SEQ ID NO:Y. Where the nucleotide position number "To" is lower than the nucleotide position number "From", the preferred ORF is the reverse complement of the referenced polynucleotide sequence. The sixth column in Table 1B.1 provides the corresponding SEQ ID NO:Y for the polypeptide sequence encoded by the preferred ORF delineated in column 5. In one embodiment, the invention provides an amino acid sequence comprising, or alternatively consisting of, a polypeptide encoded by the portion of SEQ ID NO:X delineated by "ORF (From-To)". Also provided are polynucleotides encoding such amino acid sequences and the complementary strand thereto. Column 7 in Table 1B.1 lists residues comprising epitopes contained in the polypeptides encoded by the preferred ORF (SEQ ID NO:Y), as predicted using the algorithm of Jameson and Wolf, (1988) Comp. Appl. Biosci. 4:181-186. The Jameson-Wolf antigenic analysis was performed using the computer program PROTEAN (Version 3.11 for the Power MacIntosh, DNASTAR, Inc., 1228 South Park Street Madison, WI). In specific embodiments, polypeptides of the invention comprise, or alternatively consist of, at least one, two, three, four, five or more of the predicted epitopes as described in Table 1B. It will be appreciated that depending on the analytical criteria used to predict antigenic determinants, the exact address of the determinant may vary slightly. Column 8 of Table 1B.1 ("Tissue Distribution") is described below in Table 1B.2 Column 5. Column 9 of Table 1B.1 ("Cytologic Band") provides the chromosomal location of polynucleotides corresponding to SEQ ID NO:X. Chromosomal location was determined by finding exact matches to EST and cDNA sequences contained in the

NCBI (National Center for Biotechnology Information) UniGene database. Each sequence in the UniGene database is assigned to a “cluster”; all of the ESTs, cDNAs, and STSs in a cluster are believed to be derived from a single gene. Chromosomal mapping data is often available for one or more sequence(s) in a UniGene cluster; this data (if consistent) is then applied to the cluster as a whole. Thus, it is possible to infer the chromosomal location of a new polynucleotide sequence by determining its identity with a mapped UniGene cluster.

A modified version of the computer program BLASTN (Altshul, et al., J. Mol. Biol. 215:403-410 (1990), and Gish, and States, Nat. Genet. 3:266-272) (1993) was used to search the UniGene database for EST or cDNA sequences that contain exact or near-exact matches to a polynucleotide sequence of the invention (the ‘Query’). A sequence from the UniGene database (the ‘Subject’) was said to be an exact match if it contained a segment of 50 nucleotides in length such that 48 of those nucleotides were in the same order as found in the Query sequence. If all of the matches that met this criteria were in the same UniGene cluster, and mapping data was available for this cluster, it is indicated in Table 1B under the heading “Cytologic Band”. Where a cluster had been further localized to a distinct cytologic band, that band is disclosed; where no banding information was available, but the gene had been localized to a single chromosome, the chromosome is disclosed.

Once a presumptive chromosomal location was determined for a polynucleotide of the invention, an associated disease locus was identified by comparison with a database of diseases which have been experimentally associated with genetic loci. The database used was the Morbid Map, derived from OMIM™ and National Center for Biotechnology Information, National Library of Medicine (Bethesda, MD) 2000;. If the putative chromosomal location of a polynucleotide of the invention (Query sequence) was associated with a disease in the Morbid Map database, an OMIM reference identification number was noted in column 10, Table 1B.1, labelled "OMIM Disease Reference(s). Table 5 is a key to the OMIM reference identification numbers (column 1), and provides a description of the associated disease in Column 2.

Table 1B.2

Column 5, in Table 1B.2, provides an expression profile and library code:count for each of the contig sequences (SEQ ID NO:X) disclosed in Table 1B, which can routinely be combined with the information provided in Table 4 and used to determine the tissues, cells, and/or cell line libraries which predominantly express the polynucleotides of the invention. The first number in Table 1B.2, column 5 (preceding the colon), represents the tissue/cell source identifier code corresponding to the code and description provided in Table 4. The second number in column 5 (following the colon) represents the number of times a sequence corresponding to the reference polynucleotide sequence was identified in the corresponding tissue/cell source. Those tissue/cell source identifier codes in which the first two letters are “AR” designate

information generated using DNA array technology. Utilizing this technology, cDNAs were amplified by PCR and then transferred, in duplicate, onto the array. Gene expression was assayed through hybridization of first strand cDNA probes to the DNA array. cDNA probes were generated from total RNA extracted from a variety of different tissues and cell lines. Probe synthesis was performed in the presence of ³³P dCTP, using oligo (dT) to prime reverse transcription. After hybridization, high stringency washing conditions were employed to remove non-specific hybrids from the array. The remaining signal, emanating from each gene target, was measured using a Phosphorimager. Gene expression was reported as Phosphor Stimulating Luminescence (PSL) which reflects the level of phosphor signal generated from the probe hybridized to each of the gene targets represented on the array. A local background signal subtraction was performed before the total signal generated from each array was used to normalize gene expression between the different hybridizations. The value presented after "[array code]:" represents the mean of the duplicate values, following background subtraction and probe normalization. One of skill in the art could routinely use this information to identify normal and/or diseased tissue(s) which show a predominant expression pattern of the corresponding polynucleotide of the invention or to identify polynucleotides which show predominant and/or specific tissue and/or cell expression.

TABLE 1B.1

Gene No:	cDNA Clone ID	Contig ID:	SEQ ID NO: X	ORF (From-To)	AA SEQ ID NO: Y	Predicted Epitopes	Tissue Distribution Library code: count (see Table IV for Library Codes)	Cytologic Band	OMIM Disease Reference(s):
1	HACBT91	789939	11	329 - 508	118	Ser-49 to Lys-59.	L0665: 5, L0743: 3, H0341: 2, L0761: 2, L0756: 2, S0356: 1, H0734: 1, S0280: 1, T0048: 1, H0271: 1, S0440: 1, H0641: 1, H0646: 1, L0770: 1, L0637: 1, L0800: 1, L0773: 1, L0648: 1, L0662: 1, L0768: 1, L0766: 1, L0649: 1, L0375: 1, L0784: 1, L0806: 1, L0655: 1, L0809: 1, H0672: 1, S0406: 1, L0747: 1, L0749: 1 and L0750: 1.		
2	HADDE71	839187	12	250 - 666	119	Pro-9 to Thr-14, Ser-37 to Trp-44, Gly-79 to Thr-85, Arg-88 to Lys-139.	L0769: 11, L0747: 9, L0809: 6, S0408: 4, L0770: 4, L0439: 4, L0752: 4, L0759: 4, L0766: 3, L0803: 3, L0666: 3, L0751: 3, L0780: 3, S0007: 2, H0619: 2, H0351: 2, H0333: 2, H0427: 2, H0052: 2, L0761: 2,		

3	HADDJ13	827273	13	347 - 439	120			L0744: 1, L0749: 1, L0786: 1, L0753: 1, L0755: 1, L0731: 1, L0758: 1, S0436: 1, S0011: 1 and S0192: 1. H0427: 1			
4	HADMA77	783049	14	992 - 1063	121			L0439: 15, S0222: 4, L0157: 4, L0769: 4, L0438: 3, L0745: 3, L0731: 3, L0758: 3, L0599: 3, H0443: 2, H0441: 2, S0010: 2, L0662: 2, L0744: 2, L0748: 2, L0750: 2, L0756: 2, L0777: 2, H0583: 1, L0005: 1, S0354: 1, H0675: 1, S0408: 1, H0619: 1, H0369: 1, H0574: 1, H0486: 1, H0390: 1, S0346: 1, H0309: 1, H0597: 1, T0003: 1, H0024: 1, S6028: 1, H0028: 1, T0006: 1, H0628: 1, H0135: 1, H0551: 1, S0438: 1, L0520: 1, L0768: 1, L0776: 1, L0559: 1, L0659: 1, L0384: 1, L0809: 1, H0144: 1, H0547: 1, L0746: 1,	3		

									L0747: 1, L0757: 1 and S0434: 1.			
5	HADMB15	847116	15	238 - 300	122				L0595: 2, L0442: 1, L0005: 1, L3653: 1, H0390: 1, H0081: 1, H0024: 1, L0770: 1, L5566: 1, L0651: 1, L0565: 1, L0439: 1, L0747: 1, L0752: 1, H0445: 1, L0592: 1 and L0599: 1.	7		
6	HAGBQ12	722205	16	171 - 236	123				L0754: 4, L0805: 2, L0777: 2, L0755: 2, S0010: 1, H0049: 1, L0163: 1, L0771: 1, L0775: 1 and L0776: 1.			
7	HAGCC87	638587	17	509 - 538	124				L0439: 4, L0519: 3, S0010: 2, T0010: 1, L0809: 1, H0682: 1, S0404: 1, S0406: 1, H0436: 1 and L0756: 1.			
8	HAGHN57	773286	18	900 - 932	125				H0521: 5, L0777: 5, S0376: 4, H0733: 3, H0156: 3, H0519: 3, H0436: 3, L0731: 3, H0656: 2, H0580: 2, H0747: 2, L3816: 2, H0036: 2, L0471: 2, H0090: 2, H0040: 2, H0551: 2, H0494: 2, S0438: 2, S0440: 2,			

									L0581: 1, L0595: 1, H0543: 1 and H0423: 1.			
9	HAGHR18	655435	19	28 - 126	126				L0717: 1 and S0346: 1.	8		
10	HAQAI92	688037	20	250 - 321	127				H0617: 5, H0606: 2, L0744: 2, L0779: 2, H0295: 1, H0100: 1, S0440: 1, H0026: 1, L0762: 1, L0504: 1, L0769: 1, L0764: 1, L0662: 1, L0649: 1, L0804: 1, L0787: 1, L0666: 1, L0663: 1, H0520: 1, L0748: 1, L0751: 1, L0752: 1 and S0436: 1.	20,4		
11	HAQBG57	837545	21	170 - 340	128		Trp-10 to Lys-18, Val-32 to Cys-38, Asp-41 to Thr-47.		H0295: 6, H0255: 2, H0392: 1, H0587: 1, H0333: 1, H0545: 1, H0328: 1, H0616: 1, S0142: 1, H0529: 1, L0659: 1, L0783: 1, L0528: 1, H0547: 1, S0136: 1, S0390: 1, L0754: 1, L0747: 1 and L0752: 1.			
12	HAQCE11	633730	22	262 - 273	129				H0295: 5 and L0438: 1.			
13	HBAGD86	838799	23	521 - 580	130				L0809: 4, L0766: 3, L0439: 3, H0624: 2, H0411: 2, L0794: 2, L0749: 2, L0756: 2, L0005: 1, L3649: 1,	2		

15	HBJAB02	837309	25	84 - 188	132	Arg-24 to Asp-31.	L0770: 1, L0769: 1, L0638: 1, L0775: 1, L0651: 1, L0527: 1, L0526: 1, L0789: 1, L0666: 1, L0665: 1, H0547: 1, H0435: 1, H0648: 1, S0330: 1, S0406: 1, H0627: 1, L0750: 1, L0780: 1, L0752: 1, L0758: 1, L0366: 1 and H0293: 1. S0434: 5, L0794: 3, H0255: 2, H0318: 2, H0251: 2, L0764: 2, L0628: 2, L0809: 2, L0665: 2, H0658: 2, S0406: 2, L0361: 2, H0265: 1, H0685: 1, H0657: 1, H0483: 1, S0420: 1, S0442: 1, S0358: 1, H0729: 1, H0734: 1, S0132: 1, S0222: 1, T0082: 1, H0150: 1, H0083: 1, S0214: 1, H0252: 1, H0628: 1, T0041: 1, S0344: 1, H0529: 1, L0520: 1, L0535: 1, L0662: 1, L0387: 1, L0375: 1, L0518: 1, L0666: 1, L0663: 1,	17		
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									H0726: 1, H0519: 1, H0670: 1, H0660: 1, L0602: 1, L0747: 1, L0777: 1, L0601: 1, S0276: 1, H0423: 1 and H0422: 1.			
16	HBMUH74	866160	26	344 - 430	133				L0754: 3, L0777: 3, L0439: 2, S0116: 1, H0341: 1, H0661: 1, H0038: 1, H0412: 1, L0761: 1, L0667: 1, L0764: 1, L0788: 1, H0435: 1, L0749: 1, L0779: 1 and L0758: 1.			
17	HBNAX40	834801	27	2497 - 2646	134				L0439: 11, H0171: 5, L0754: 5, L0748: 4, H0052: 3, L0662: 3, L0756: 3, L0755: 3, H0422: 3, S0360: 2, L0738: 2, H0032: 2, L0803: 2, L0655: 2, L0789: 2, L0605: 2, H0423: 2, H0638: 1, T0114: 1, H0156: 1, L0021: 1, S0010: 1, H0581: 1, H0046: 1, L0471: 1, H0014: 1, H0356: 1, H0188: 1, H0553: 1, H0591: 1, S0386: 1, T0042: 1, H0625: 1, H0641: 1,			

									S0142: 1, L0598: 1, L0369: 1, L0640: 1, L0375: 1, L0654: 1, L0659: 1, L0783: 1, L0663: 1, L0665: 1, H0144: 1, L0352: 1, H0547: 1, H0648: 1, H0672: 1, H0555: 1, H0436: 1, L0749: 1, L0779: 1, L0731: 1, L0758: 1, L0759: 1, H0445: 1, L0366: 1 and H0668: 1.			
18	HBXCX15	637542	28	72 - 77	135				S0038: 3, H0438: 1, L0363: 1 and S0053: 1.			
19	HCDBO32	831942	29	1669 - 1884	136	Val-2 to Thr-7.			L0803: 7, L0766: 4, L0777: 4, L0666: 3, H0521: 3, T0115: 2, H0687: 2, L0809: 2, H0659: 2, L0754: 2, L0779: 2, L0759: 2, L3643: 1, H0341: 1, H0747: 1, H0749: 1, L3387: 1, H0351: 1, S0222: 1, H0441: 1, L3816: 1, H0013: 1, S0280: 1, H0251: 1, H0544: 1, H0123: 1, H0354: 1, H0266: 1, H0622: 1, H0090: 1, T0041: 1, H0641: 1,			

									S0422: 1, L0371: 1, L0646: 1, L0662: 1, L0774: 1, L0805: 1, L0653: 1, L0659: 1, L0635: 1, L0526: 1, L0783: 1, L0663: 1, L0664: 1, L0665: 1, H0144: 1, T0068: 1, L3811: 1, H0519: 1, H0682: 1, S0152: 1, S0136: 1, L0744: 1, L0780: 1, L0758: 1, H0444: 1, H0445: 1, L0590: 1, L0594: 1, S0026: 1 and H0422: 1.			
20	HCEEE79	560609	30	131 - 298	137	Gly-35 to Pro-41.			H0052: 1			
21	HCEFZ82	831745	31	215 - 1012	138	Tyr-30 to Gln-35, Asn-114 to Lys-119, Ser-161 to Ala-171, Arg-183 to Gly-189, Pro-205 to Ala-211, Lys-231 to Trp-237, Gly-246 to Lys-265.			L0748: 11, H0052: 8, L0803: 8, L0749: 8, L0770: 7, L0439: 5, L0746: 4, L0752: 4, L3811: 3, H0575: 2, H0012: 2, H0031: 2, L0768: 2, L0804: 2, L0774: 2, L0740: 2, L0747: 2, L0756: 2, L0779: 2, L0757: 2, L0758: 2, L0592: 2, L0593: 2, H0556: 1, S0420: 1, S0376: 1, H0441: 1, H0632: 1, S0010: 1, T0115: 1,			

								H0545: 1, H0009: 1, H0620: 1, H0197: 1, H0051: 1, S0388: 1, S0051: 1, H0252: 1, H0032: 1, L0455: 1, H0591: 1, H0272: 1, L0564: 1, S0438: 1, S0344: 1, L0373: 1, L0646: 1, L0794: 1, L0766: 1, L0805: 1, L0776: 1, L0783: 1, L0809: 1, S0374: 1, H0522: 1, H0134: 1, L0780: 1, L0731: 1, L0759: 1, S0436: 1, L0597: 1, H0543: 1, H0423: 1 and L0600: 1.			
22	HCUCF89	637986	32	189 - 278	139	Gly-14 to Asp-21.	H0306: 1, L0761: 1 and H0436: 1.				
23	HCWAE64	535893	33	410 - 427	140		H0305: 1	9			
24	HCWUL09	834722	34	333 - 368	141		H0305: 9, H0589: 2 and 6 S0001: 1.				
25	HDPDI72	897277	35	23 - 385	142	Arg-63 to Phe-72, Ile-114 to Phe-120.	H0521: 2 and H0580: 1.	10			
26	HDPFY18	779450	36	161 - 184	143		S0114: 1, H0427: 1, H0123: 1, H0688: 1, H0264: 1, L0547: 1, L0518: 1, L3811: 1, H0521: 1, H0445: 1 and H0543: 1.	11			
27	HDPJE44	899328	37	169 - 351	144		L3811: 7, L0439: 7,				

						L0794: 6, L0759: 5, L0591: 5, L0803: 4, L0805: 4, L2653: 4, H0547: 4, L0748: 4, L0755: 4, L0596: 4, H0171: 3, L2886: 3, S0376: 3, S0007: 3, H0024: 3, H0355: 3, H0615: 3, H0428: 3, H0090: 3, H0623: 3, S0422: 3, L0766: 3, L0659: 3, H0144: 3, H0658: 3, S0406: 3, L0749: 3, L0758: 3, S0436: 3, H0624: 2, H0717: 2, S0358: 2, S0360: 2, H0486: 2, L2636: 2, H0427: 2, S0010: 2, H0052: 2, H0251: 2, H0687: 2, H0622: 2, H0553: 2, H0644: 2, H0591: 2, S0438: 2, L0770: 2, L0769: 2, L0662: 2, S0374: 2, L3827: 2, L3828: 2, S0126: 2, H0689: 2, H0670: 2, H0521: 2, S0028: 2, L0744: 2, L0740: 2, L0754: 2, L0752: 2, L0593: 2, S0192: 2,		
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					S0312: 2, H0551: 2, L3815: 2, L0764: 2, L0663: 2, H0144: 2, L3825: 2, L0751: 2, L0754: 2, L0745: 2, L0731: 2, L0589: 2, H0653: 2, H0136: 2, H0216: 2, H0624: 1, S6024: 1, S0430: 1, H0656: 1, H0255: 1, S0046: 1, H0747: 1, H0645: 1, L2759: 1, H0013: 1, H0156: 1, H0575: 1, H0050: 1, S0050: 1, H0373: 1, H0687: 1, S0314: 1, S0250: 1, H0031: 1, H0135: 1, H0634: 1, H0616: 1, H0380: 1, H0264: 1, H0433: 1, H0059: 1, L0351: 1, S0422: 1, L0800: 1, L0662: 1, L0626: 1, L0766: 1, L0803: 1, L0375: 1, L0655: 1, L0659: 1, L0783: 1, L0809: 1, L0664: 1, L2263: 1, L2258: 1, L2259: 1, H0726: 1, L3826: 1, L3827: 1, H0648: 1, S0152: 1,	
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31	HE6CS65	762960	41	295 - 483	148	Trp-50 to Leu-55.	L0777: 16, L0748: 12, L0757: 11, L0776: 8, L0439: 7, H0692: 6, H0046: 6, L0769: 5, L0666: 5, S0242: 5, L0770: 4, L0771: 4, L0438: 4, L0743: 4, L0754: 4, L0749: 4, L0758: 4, S0444: 3, H0051: 3, L0662: 3, L0766: 3, S0378: 3, L0751: 3, L0747: 3, S0436: 3, S0212: 2, H0637: 2, H0497: 2, H0545: 2, H0050: 2, H0031: 2, H0090: 2, H0100: 2, L0768: 2, L0561: 2, L0774: 2, L0775: 2, L0657: 2, H0670: 2, S3014: 2, L0744: 2, L0752: 2, L0581: 2, H0624: 1, H0170: 1, H0713: 1, H0717: 1, S6024: 1, T0049: 1, H0255: 1, S0356: 1, S0442: 1, S0358: 1, S0376: 1, S0360: 1, H0619: 1, L3651: 1, L0717: 1, S0278: 1, H0391: 1, H0333: 1, H0013: 1,		
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									H0053: 1, H0575: 1, S0346: 1, H0052: 1, H0263: 1, H0596: 1, L0738: 1, H0572: 1, H0510: 1, H0266: 1, H0688: 1, H0039: 1, H0622: 1, H0111: 1, H0181: 1, H0617: 1, H0032: 1, H0169: 1, H0634: 1, H0087: 1, H0412: 1, S0450: 1, S0440: 1, L0639: 1, L0637: 1, L0372: 1, L0646: 1, L0651: 1, L0806: 1, L0659: 1, L0792: 1, L0664: 1, L0665: 1, S0216: 1, H0144: 1, H0697: 1, S0374: 1, L3812: 1, H0520: 1, H0547: 1, H0658: 1, H0660: 1, H0648: 1, H0521: 1, H0696: 1, S0027: 1, S0028: 1, L0741: 1, L0740: 1, L0779: 1, L0731: 1, L0759: 1, S0260: 1, H0445: 1, S0434: 1, L0362: 1 and L0366: 1.				
32	HE8BQ49	589443	42	133 - 168	149				H0013: 2				
33	HE9CY05	834826	43	55 - 762	150	Ser-18 to Glu-24,			L0748: 8, L0749: 3,				

						Leu-121 to Asp-134, Pro-142 to Ala-154, Cys-185 to Val-203.	L0471: 2 and H0144: 1.		
34	HEAAW94	847340	44	189 - 224	151		L0439: 26, L0438: 20, L0748: 17, L0766: 16, L0754: 16, L0731: 16, H0556: 9, L0740: 8, S0222: 7, H0090: 7, L0774: 7, H0144: 6, L0745: 6, L0779: 6, L0777: 6, L0758: 6, S0003: 5, L0662: 5, L0794: 5, S0418: 4, H0575: 4, L0776: 4, L0751: 4, L0749: 4, L0756: 4, L0780: 4, L0752: 4, L0591: 4, H0423: 4, H0341: 3, S0360: 3, H0369: 3, H0156: 3, L0435: 3, L0769: 3, L0775: 3, L0809: 3, L0666: 3, H0547: 3, S0328: 3, H0521: 3, L0747: 3, L0750: 3, H0543: 3, H0171: 2, S0442: 2, S0354: 2, S0358: 2, S0132: 2, S0278: 2, H0497: 2, T0039: 2, H0706: 2, H0036: 2, S0474: 2, H0596: 2,		

35	HEBFR46	847064	45	200 - 289	152	Met-1 to Thr-6.	H0520: 1, H0519: 1, S0126: 1, H0672: 1, H0754: 1, S0152: 1, H0522: 1, H0696: 1, S0044: 1, S0406: 1, L0612: 1, S3012: 1, L0746: 1, L0786: 1, L0759: 1, H0445: 1, L0684: 1, L0608: 1, H0667: 1, S0276: 1 and H0422: 1.		
							H0457: 10, H0550: 5, H0436: 5, H0549: 4, H0616: 4, L0519: 4, H0556: 3, H0580: 3, S0007: 3, S0046: 3, L0809: 3, L0747: 3, L0777: 3, S0436: 3, H0295: 2, T0040: 2, H0266: 2, L0761: 2, L0783: 2, L0789: 2, H0658: 2, H0521: 2, L0753: 2, L0731: 2, L0596: 2, H0543: 2, S0040: 1, S0116: 1, S0282: 1, H0662: 1, H0402: 1, H0125: 1, L0534: 1, L0562: 1, S0356: 1, S0358: 1, H0749: 1, L3816: 1, H0559: 1, H0069: 1,		7

									H0599: 1, H0618: 1, H0253: 1, H0581: 1, H0546: 1, H0123: 1, S0051: 1, H0083: 1, H0687: 1, H0284: 1, H0124: 1, H0038: 1, H0551: 1, H0623: 1, S0038: 1, T0041: 1, S0440: 1, S0150: 1, L3818: 1, S0002: 1, L0763: 1, L0769: 1, L5575: 1, L0627: 1, L0800: 1, L0662: 1, L0803: 1, L0793: 1, L0666: 1, L2264: 1, L3825: 1, L3827: 1, L3828: 1, H0547: 1, H0519: 1, H0539: 1, S0037: 1, S0206: 1, L0748: 1, L0749: 1, H0595: 1, L0593: 1, S0194: 1 and S0276: 1.				
36	HEOMC46	866171	46	154 - 309	153	Ser-5 to Thr-10, Cys-36 to Glu-51.	H0749: 2, H0581: 2, H0457: 2 and S0116: 1.						
37	HFCDW95	847383	47	151 - 159	154		L0766: 9, L0803: 8, H0341: 7, H0521: 7, L0770: 6, L0771: 6, L0754: 6, L0752: 6, L0731: 6, S0354: 5, S0422: 5, L0662: 5, H0519: 5, L0439: 5,	7					

39	HFIJA29	839206	49	175 - 423	156	Ser-36 to Ser-42, Lys-54 to Ser-69.	S0328: 1, H0436: 1, L0749: 1, L0750: 1, L0777: 1, L0752: 1, L0599: 1, S0242: 1 and H0422: 1.		
							L0766: 20, L0754: 10, L0776: 8, L0803: 5, L0749: 5, H0661: 4, L0740: 4, L0751: 4, L0608: 4, L0770: 3, L0750: 3, L0761: 2, L0794: 2, L0806: 2, L0783: 2, L0809: 2, L0789: 2, L0438: 2, S0404: 2, L0745: 2, L0777: 2, L0755: 2, L0758: 2, S0134: 1, H0638: 1, S0358: 1, S0408: 1, S0045: 1, S0046: 1, H0581: 1, H0023: 1, H0355: 1, S0214: 1, L0055: 1, H0477: 1, L0796: 1, L3905: 1, L0772: 1, L0646: 1, L0800: 1, L0642: 1, L0764: 1, L0773: 1, L0363: 1, L0768: 1, L0804: 1, L0774: 1, L0805: 1, L0655: 1, L0807: 1, L0526: 1, L0531: 1,	6,7	

									H0689: 1, S0378: 1, S0152: 1, S0406: 1, H0732: 1, L0742: 1, L0748: 1, L0747: 1, L0753: 1, L0757: 1, S0194: 1, H0422: 1 and S0424: 1.						
40	HFKFX64	566835	50	127 - 171	157				H0012: 3 and L0809: 1. 18,N/A						
41	HGBER72	826710	51	43 - 102	158				L0766: 12, H0436: 9, H0543: 8, L0769: 6, L0749: 6, L0731: 6, H0556: 5, L0655: 5, S0434: 5, L0439: 4, L0758: 4, S0114: 3, H0255: 3, L3904: 3, L0794: 3, L0776: 3, L0659: 3, L0783: 3, L0809: 3, L0751: 3, H0423: 3, S0358: 2, S0360: 2, S0007: 2, H0549: 2, H0550: 2, H0486: 2, H0014: 2, S0388: 2, H0424: 2, H0031: 2, H0628: 2, L5575: 2, L0771: 2, L0662: 2, L0791: 2, L0793: 2, L2265: 2, L0438: 2, S0328: 2, L0740: 2, L0756: 2, H0265: 1, H0686: 1, S0134: 1, H0657: 1,						

					H0656: 1, S0001: 1, S0418: 1, L0619: 1, S0442: 1, S0408: 1, H0730: 1, H0749: 1, H0619: 1, H0351: 1, S0222: 1, H0592: 1, H0586: 1, T0060: 1, H0250: 1, H0618: 1, H0318: 1, H0052: 1, H0251: 1, H0545: 1, H0569: 1, H0012: 1, H0201: 1, S6028: 1, H0288: 1, H0622: 1, T0023: 1, L0483: 1, H0604: 1, S0036: 1, H0135: 1, H0040: 1, H0264: 1, S0039: 1, L0640: 1, L0763: 1, L0770: 1, L0761: 1, L0648: 1, L0521: 1, L0533: 1, L0774: 1, L0775: 1, L0376: 1, L0378: 1, L0629: 1, L5623: 1, L0666: 1, L0664: 1, S0310: 1, L3811: 1, H0689: 1, H0659: 1, H0660: 1, H0648: 1, H0696: 1, H0576: 1, S0028: 1, L0742: 1, L0750: 1, L0779: 1, L0777: 1,	
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42	HGBGN34	648659	52	280 - 426	159	Asn-2 to Val-8.	L0752: 1, L0591: 1, L0601: 1, H0542: 1 and H0506: 1. L0747: 5, H0716: 2, H0427: 2, S0280: 2, H0662: 1, S0444: 1, H0441: 1, H0492: 1, T0001: 1, H0014: 1, H0030: 1, H0674: 1, L5575: 1, L0659: 1, S0330: 1, L0752: 1 and S0436: 1.			
43	HGLBG15	701990	53	191 - 271	160		L0803: 19, S0474: 17, L0748: 13, S0408: 11, H0351: 11, L2669: 11, L2504: 10, L0770: 10, L0805: 9, L0439: 9, L0754: 9, S0422: 8, L0809: 8, L0794: 6, L0755: 6, L0731: 6, L0758: 6, S0360: 5, H0265: 4, S0414: 4, H0581: 4, H0271: 4, L0771: 4, L0804: 4, L0776: 4, L0659: 4, L0666: 4, L0749: 4, L0591: 4, H0327: 3, L0806: 3, L0655: 3, L0636: 3, L0565: 3, H0436: 3, L0777: 3, S0434: 3, S0436: 3,	7		

44	HHFEC39	609873	54	1211 - 1216	161			H0710: 1, H0521: 1, H0522: 1, H0696: 1, S0044: 1, S0406: 1, S0027: 1, L0742: 1, L0744: 1, L0751: 1, L0745: 1, L0747: 1, L0779: 1, L0780: 1, L0757: 1, L0759: 1, S0031: 1, S0260: 1, L0596: 1, L0605: 1, L0595: 1, S0026: 1, S0192: 1, S0242: 1, H0542: 1, H0543: 1, S0042: 1 and S0462: 1.			
								L0805: 21, L0776: 19, L0751: 14, L0759: 13, L0770: 11, H0615: 8, L0803: 8, L0438: 7, L0439: 7, L0758: 7, L0769: 6, L0521: 6, L0754: 6, H0624: 5, H0486: 5, L0500: 5, L0807: 5, L0740: 5, L0591: 5, H0716: 4, H0351: 4, L0774: 4, H0144: 4, S0328: 4, L0748: 4, L0745: 4, L0604: 4, S0414: 3, H0013: 3, S0250: 3, H0428: 3, H0644: 3, H0591: 3, L0659: 3,			

45	HHSDI53	862028	55	221 - 295	162				H0510: 1, H0271: 1, H0328: 1, H0039: 1, H0622: 1, L0483: 1, H0124: 1, S0036: 1, H0038: 1, H0616: 1, H0412: 1, H0059: 1, H0100: 1, H0646: 1, S0422: 1, S0002: 1, H0743: 1, H0529: 1, L0520: 1, L0640: 1, L0763: 1, L0773: 1, L0364: 1, L0649: 1, L0497: 1, L0526: 1, L0788: 1, L0789: 1, L0663: 1, L0665: 1, S0374: 1, H0780: 1, H0547: 1, S0126: 1, H0689: 1, H0648: 1, S0330: 1, S0378: 1, H0522: 1, S0037: 1, L0747: 1, L0750: 1, L0780: 1, L0752: 1, H0595: 1, S0434: 1, L0608: 1, L0594: 1, L0361: 1, L0603: 1, S0026: 1, S0192: 1, S0194: 1, H0423: 1, S0398: 1, S0460: 1, H0506: 1, H0008: 1 and H0352: 1.		
									L0766: 10, L0752: 8, 1,18		

									L0528: 1, L5622: 1, L0790: 1, H0547: 1, H0648: 1, H0539: 1, S0152: 1, H0696: 1, S0044: 1, S0406: 1, S0028: 1, L0758: 1, S0434: 1, S0436: 1, L0366: 1, S0011: 1, S0276: 1, H0422: 1, S0398: 1 and S0424: 1.			
46	HISBA38	561711	56	169 - 279	163				L0766: 3, H0318: 1 and 9 H0539: 1.			
47	HJPBK28	638191	57	256 - 387	164				L0794: 6, L0439: 5, L0759: 5, H0556: 4, L0771: 4, L0770: 3, L0643: 3, H0144: 3, H0156: 2, H0188: 2, H0090: 2, H0641: 2, L0662: 2, L0766: 2, L0803: 2, L0776: 2, L0661: 2, L0659: 2, L0790: 2, H0522: 2, S0436: 2, H0295: 1, T0049: 1, H0583: 1, S0116: 1, H0663: 1, H0662: 1, S0356: 1, S0376: 1, S0132: 1, H0586: 1, H0587: 1, H0486: 1, H0575: 1, H0748: 1, H0744: 1, H0309: 1, H0231: 1,			

									H0331: 1, H0559: 1, T0039: 1, H0013: 1, S0280: 1, H0318: 1, T0110: 1, H0024: 1, S0364: 1, H0591: 1, H0038: 1, H0040: 1, S0142: 1, L0640: 1, L0667: 1, L0764: 1, L0662: 1, L0804: 1, L0659: 1, L0517: 1, L0789: 1, L4559: 1, L0664: 1, S0126: 1, H0435: 1, H0539: 1, S0152: 1, H0521: 1, H0522: 1, S0027: 1, L0779: 1, L0758: 1, L0485: 1, L0601: 1, S0026: 1, H0667: 1, S0192: 1, H0542: 1 and H0506: 1.				
49	HL YGE16	651339	59	406 - 627	166	Arg-23 to Trp-42, Val-52 to Pro-61.	H0255: 5, H0144: 3, H0429: 2, L0662: 2, L0794: 2, L0803: 2, L0809: 2, L0758: 2, L0599: 2, H0542: 2, S0040: 1, H0650: 1, S0442: 1, H0642: 1, L0157: 1, H0571: 1, H0673: 1, H0494: 1, L0771: 1, L0766: 1, L0776: 1, L0629: 1,	7					

									L0657: 1, L0659: 1, L0792: 1, L0565: 1, H0345: 1, L0748: 1, L0754: 1, L0747: 1, L0749: 1, H0445: 1 and S0242: 1.			
50	HLYGY91	658703	60	211 - 339	167				H0692: 10, L0777: 10, L0805: 5, L0803: 3, L2497: 2, H0328: 2, L0662: 2, L0794: 2, L0809: 2, L3832: 2, L0748: 2, L0752: 2, L0599: 2, H0170: 1, H0402: 1, S0444: 1, S0360: 1, H0747: 1, L2486: 1, L3503: 1, H0427: 1, H0644: 1, H0038: 1, L0800: 1, L0648: 1, L0804: 1, H0670: 1, H0478: 1, L0731: 1, L0758: 1, H0445: 1, S0434: 1, L0591: 1 and L0362: 1.			
51	HMCFH60	654853	61	211 - 357	168				L0659: 10, T0040: 9, L0665: 9, L0759: 9, L0519: 8, L0776: 7, S0436: 7, L0744: 6, L0747: 6, L0749: 6, L0758: 6, S0418: 5, H0052: 5, H0457: 5, H0150: 5, L0769: 5,	6		

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									S0126: 1, H0682: 1, H0659: 1, S0328: 1, S0380: 1, H0710: 1, H0521: 1, H0522: 1, H0627: 1, S0028: 1, L0741: 1, L0742: 1, L0439: 1, L0740: 1, L0756: 1, L0786: 1, L0780: 1, L0755: 1, L0581: 1, L0595: 1, L0601: 1, H0667: 1, S0192: 1, H0542: 1, L0718: 1 and S0424: 1.			
52	HMIK10	562774	62	195 - 290	169				S6028: 1	11		
53	HMIBD93	634227	63	983 - 1180	170	Pro-4 to Gly-13, Ala-42 to Ser-50.			L0439: 6, L0751: 5, L0770: 3, L0769: 3, L0764: 3, H0617: 2, L0766: 2, L0752: 2, H0445: 2, S6024: 1, H0351: 1, S0222: 1, H0586: 1, S0010: 1, S6028: 1, L0768: 1, L0794: 1, L0438: 1, L0747: 1, L0753: 1 and L0758: 1.	2		
54	HMWJF53	758158	64	1015 - 1131	171				H0255: 7, H0318: 5, H0620: 5, L0754: 5, L0766: 4, L0666: 4, S0358: 3, H0457: 3, H0135: 3, L0776: 3, L0809: 3, H0696: 3,	2		

						S3012: 3, H0624: 2, H0295: 2, H0254: 2, H0662: 2, H0402: 2, H0305: 2, S0132: 2, L0717: 2, L0021: 2, H0617: 2, H0673: 2, L0769: 2, L0638: 2, L0796: 2, L0667: 2, L0662: 2, L0653: 2, L0783: 2, L0663: 2, S0126: 2, H0539: 2, H0521: 2, S0044: 2, S0027: 2, L0745: 2, L0747: 2, L0755: 2, L0587: 2, H0352: 2, H0170: 1, H0556: 1, H0657: 1, H0341: 1, S0212: 1, S0418: 1, S0360: 1, S0410: 1, H0339: 1, H0549: 1, S0222: 1, H0441: 1, H0331: 1, H0486: 1, H0427: 1, H0575: 1, T0048: 1, H0581: 1, H0052: 1, H0545: 1, H0150: 1, H0570: 1, H0569: 1, L0163: 1, H0083: 1, H0355: 1, H0252: 1, H0039: 1, T0023: 1, H0124: 1, H0090: 1, H0413: 1,					
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									H0560: 1, H0561: 1, S0372: 1, H0509: 1, H0652: 1, S0144: 1, S0422: 1, L0762: 1, L0770: 1, L0761: 1, L0373: 1, L0372: 1, L0645: 1, L0764: 1, L0771: 1, L0648: 1, L0768: 1, L0649: 1, L0804: 1, L0651: 1, L0806: 1, L0655: 1, L0659: 1, L0517: 1, L0528: 1, L0665: 1, H0698: 1, S0374: 1, L0438: 1, H0684: 1, H0658: 1, H0670: 1, S0328: 1, S0380: 1, H0134: 1, S0406: 1, L0743: 1, L0749: 1, L0750: 1, L0779: 1, L0759: 1, S0031: 1, H0445: 1, H0653: 1, S0194: 1, S0276: 1, H0542: 1 and S0460: 1.					
55	HNECL22	799541	65	472 - 576	172				L0748: 54, L0766: 20, L0754: 18, H0179: 12, L0777: 12, L0750: 11, L0749: 10, S0116: 9, H0271: 9, L0761: 9, H0031: 8, L0794: 8, H0144: 8, L0744: 8,	8				

									H0056: 1, H0623: 1, T0041: 1, T0042: 1, S0210: 1, S0002: 1, S0426: 1, L0598: 1, L0641: 1, L0764: 1, L0768: 1, L0807: 1, L0514: 1, L0658: 1, L0783: 1, L5623: 1, L0788: 1, L0663: 1, L0665: 1, S0374: 1, H0519: 1, S0122: 1, H0659: 1, H0658: 1, H0666: 1, H0672: 1, S0328: 1, H0521: 1, H0522: 1, S0406: 1, H0555: 1, H0478: 1, H0727: 1, L0742: 1, L0755: 1, L0731: 1, S0011: 1, S0026: 1, H0543: 1, H0423: 1, H0422: 1 and H0506: 1.		
56	HNFAC50	815676	66	676 - 774	173	Lys-7 to Glu-18.	L0769: 5, L0756: 4, S0444: 3, L0774: 3, H0624: 2, S0408: 2, H0587: 2, L0764: 2, L0766: 2, H0170: 1, H0497: 1, H0333: 1, H0156: 1, L0022: 1, H0271: 1, S0344: 1, L0637: 1, L0772: 1, L0773: 1, L0662: 1,				

									L0775: 1, L0809: 1, L0791: 1, L0663: 1, H0144: 1, S0374: 1, L3811: 1, H0593: 1, H0660: 1, H0648: 1, H0672: 1, H0696: 1, L0749: 1, L0750: 1, L0779: 1, L0752: 1, L0755: 1, L0599: 1, L0601: 1 and H0667: 1.			
57	HNGEA34	815678	67	58 - 192	174	His-26 to Ser-32.			H0393: 1 and S0052: 1. 2			
58	HNGIV64	561572	68	221 - 247	175				S0052: 1			
59	HNGKT41	836061	69	415 - 552	176				S0428: 1	5		
60	HNGNO53	836063	70	467 - 571	177				S0428: 2 and L0439: 1. 10,8			
61	HNHCT47	634691	71	73 - 192	178	Asn-25 to Thr-33.			S0053: 2 and S0046: 1. 11,17,5			
62	HNHKI74	777856	72	127 - 159	179				S0216: 1			
63	HORBS82	638293	73	21 - 140	180	Gly-30 to Ser-35.			H0706: 2, L0809: 2, S0360: 1, L0623: 1, H0122: 1, H0041: 1, H0095: 1, H0292: 1, H0424: 1, S0364: 1, L0794: 1, L0787: 1, L0663: 1, H0780: 1, H0435: 1, L0743: 1, L0747: 1 and L0731: 1.	20		
64	HOUDE92	580866	74	70 - 336	181	Pro-22 to His-31, Ser-80 to Gln-88.			H0052: 17, L0745: 11, L0748: 10, H0547: 7, L0439: 7, L0755: 6, L0771: 5, L0774: 5, L0662: 4, L0746: 4, L0777: 4, S0474: 3,	12p13		

									L2260: 1, L2381: 1, L2673: 1, L3827: 1, H0520: 1, S0126: 1, L3832: 1, L0753: 1, S0434: 1, L0599: 1, S0011: 1, H0667: 1, L3560: 1 and L3585: 1.			
67	HPCAL26	762822	77	1021 - 1113	184				L0659: 11, S0126: 11, L0731: 11, S0192: 11, L0666: 9, L0777: 7, T0049: 5, S0358: 5, L0771: 5, L0757: 5, S0360: 4, S0440: 4, L0740: 4, L0758: 4, S0212: 3, S0356: 3, S0046: 3, H0369: 3, H0545: 3, L0662: 3, L0774: 3, L0809: 3, H0519: 3, L0752: 3, S0011: 3, H0295: 2, H0662: 2, S0468: 2, H0012: 2, H0024: 2, H0356: 2, H0616: 2, H0268: 2, H0412: 2, L0646: 2, L0803: 2, S0013: 2, L0754: 2, L0747: 2, L0759: 2, S0040: 1, S0418: 1, S0442: 1, S0376: 1, H0676: 1, L0717: 1, H0550: 1, S0222: 1,	11		

									H0383: 1, H0212: 1, H0388: 1, H0135: 1, H0040: 1, H0487: 1, H0413: 1, T0069: 1, H0560: 1, H0538: 1, S0210: 1, L0763: 1, L0646: 1, L0641: 1, L0649: 1, L0803: 1, L0652: 1, L0629: 1, L0659: 1, L0787: 1, L0665: 1, H0435: 1, H0528: 1, H0521: 1, H0555: 1, L0779: 1, L0581: 1, S0276: 1 and H0008: 1.			
72	HSQBF66	560726	82	229 - 429	189				S0026: 1			
73	HSRFD18	840771	83	67 - 153	190				L0754: 10, S0422: 5, S0022: 4, L0803: 4, L0748: 4, L0747: 4, L0591: 4, H0486: 3, L0766: 3, L0805: 3, L0526: 3, L0665: 3, S0434: 3, S0212: 2, S0444: 2, S0360: 2, S0222: 2, L3816: 2, H0013: 2, H0596: 2, L0471: 2, H0166: 2, H0591: 2, H0509: 2, L0646: 2, L0662: 2, L0659: 2, L0666: 2, L0664: 2, S0374: 2,	1		

									L0779: 2, L0777: 2, L0759: 2, S0436: 2, H0624: 1, H0170: 1, S0114: 1, S0001: 1, H0671: 1, H0663: 1, H0402: 1, H0305: 1, S0442: 1, S0408: 1, H0329: 1, H0742: 1, L3387: 1, H0581: 1, H0421: 1, H0194: 1, H0263: 1, H0597: 1, H0569: 1, H0355: 1, H0510: 1, H0179: 1, H0687: 1, H0615: 1, L0483: 1, H0553: 1, H0644: 1, H0673: 1, H0674: 1, H0100: 1, S0450: 1, H0714: 1, L0763: 1, L0770: 1, L0761: 1, L0649: 1, L0776: 1, L0518: 1, L0790: 1, L0791: 1, L0792: 1, L0663: 1, H0547: 1, H0670: 1, H0521: 1, H0696: 1, S0406: 1, H0555: 1, H0478: 1, L0780: 1, H0707: 1, S0276: 1 and H0543: 1.						
74	HSWBE76	751308	84	380 - 559	191				L0777: 4, L0751: 3, L0747: 3, L0648: 2,						

								L0779: 2, L0753: 2, S0342: 1, H0484: 1, H0661: 1, S0358: 1, L0009: 1, H0411: 1, S6014: 1, H0546: 1, H0123: 1, H0188: 1, S0366: 1, H0413: 1, S0344: 1, H0529: 1, L0769: 1, L0627: 1, L0774: 1, L0378: 1, L0776: 1, L0655: 1, L0663: 1, S0380: 1, H0478: 1, L0743: 1, L0750: 1 and S0196: 1.			
75	HT3BF49	838620	85	306 - 320	192			H0271: 2, L0791: 2, L0439: 2, H0159: 1, H0561: 1, L0774: 1, S0052: 1 and L0779: 1.	6		
76	HTEEW69	764835	86	182 - 1153	193	Asp-63 to Thr-70, Asn-77 to Ser-86, Thr-101 to Arg-108, Pro-117 to Asn-123, Gly-194 to Trp-203.		H0038: 8, H0616: 4, L0779: 3, L0758: 3, L0753: 2, L0032: 1, T0006: 1, H0040: 1, L0768: 1 and H0547: 1.			
77	HTEHU59	840385	87	170 - 274	194	Ser-29 to Phe-34.		S0422: 6, H0038: 4, L0758: 4, L0754: 3, S0360: 2, H0024: 2, L0598: 2, L0766: 2, L0748: 2, L0747: 2, L0756: 2, H0583: 1, H0341: 1, S0418: 1, L0005: 1, H0741: 1,	11		

79	HTGBK95	834490	89	271 - 321	196				L0608: 1 and H0542: 1. L0777: 5, S0444: 3, L0766: 3, L0803: 3, L0439: 3, S0360: 2, L0598: 2, L0666: 2, L0748: 2, T0049: 1, S0134: 1, S0116: 1, S0408: 1, L0717: 1, H0586: 1, H0486: 1, H0575: 1, H0510: 1, H0553: 1, H0560: 1, S0422: 1, L0763: 1, L0769: 1, L0521: 1, L0767: 1, L0768: 1, L0775: 1, L0663: 1, S0374: 1, L0438: 1, H0520: 1, H0682: 1, S0328: 1, S0406: 1, L0740: 1, S0192: 1 and H0543: 1.			
80	HTLEM16	779133	90	1220 - 1429	197	Arg-29 to Cys-43.			L0439: 31, L0741: 24, H0056: 13, L0748: 12, H0052: 9, H0521: 9, L0776: 8, L0744: 8, L0438: 7, L0754: 7, S0474: 6, L0766: 6, L0742: 6, L0731: 6, L0750: 5, S0278: 4, L5566: 4, L0665: 4, H0522: 4, H0556: 3, H0716: 3, H0657: 3,			

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82	HTODN35	570901	92	67 - 111	199			H0264: 1			
83	HTPDU17	840596	93	52 - 153	200			H0677: 19, L0759: 6, L0748: 5, H0040: 4, L0438: 3, L0754: 3, L0750: 3, L0777: 3, H0255: 2, H0617: 2, H0038: 2, H0529: 2, L0769: 2, L0761: 2, L0662: 2, L0666: 2, S0406: 2, L0749: 2, L0758: 2, L0595: 2, H0265: 1, H0556: 1, H0717: 1, S0134: 1, H0650: 1, H0657: 1, S0358: 1, S0444: 1, S0410: 1, S0045: 1, H0411: 1, H0392: 1,			

									L0468: 1, H0587: 1, H0013: 1, H0069: 1, H0635: 1, H0575: 1, H0618: 1, H0581: 1, H0564: 1, H0569: 1, S6028: 1, H0266: 1, H0252: 1, H0615: 1, H0039: 1, H0031: 1, H0634: 1, H0100: 1, H0494: 1, H0334: 1, H0561: 1, S0150: 1, S0422: 1, L0667: 1, L0646: 1, L0800: 1, L0771: 1, L0661: 1, L0809: 1, L0790: 1, L0792: 1, L0663: 1, L0665: 1, S0374: 1, H0547: 1, H0519: 1, H0593: 1, H0672: 1, H0518: 1, H0521: 1, H0555: 1, H0436: 1, L0439: 1, L0779: 1, L0731: 1 and L0757: 1.			
84	HTTDN24	766485	94	1024 - 1728	201	Asp-194 to Leu-199, Ile-206 to Pro-211, Glu-224 to Ser-229.						
85	HTTEE41	840950	95	1171 - 1197	202		H0040: 17, H0251: 14, L0758: 10, L0748: 8, L0731: 8, H0494: 7, L0666: 7, H0144: 7, H0659: 7, L0747: 7,	12				

								H0665: 1, S0196: 1, L2368: 1, H0008: 1 and H0352: 1.			
86	HTXJD85	840391	96	211 - 306	203			H0556: 2, L0638: 1, L0748: 1 and L0439: 1.	3		
87	HUVDJ48	564853	97	196 - 213	204			H0393: 1, H0056: 1 and L0662: 1.			
88	HWBBU75	780360	98	783 - 938	205	Ser-17 to Gly-22, Leu-34 to Ala-42.		L0665: 4, H0457: 3, H0264: 3, L0766: 3, H0521: 3, L0745: 3, H0556: 2, H0580: 2, S0352: 2, L0761: 2, L0806: 2, L0789: 2, L0748: 2, H0542: 2, H0255: 1, S0278: 1, H0581: 1, H0271: 1, H0719: 1, H0413: 1, H0494: 1, S0002: 1, S0426: 1, L0769: 1, L0774: 1, H0660: 1, L0750: 1, L0752: 1, L0753: 1 and S0424: 1.			
89	HWHPB78	740778	99	200 - 400	206	Gln-25 to Leu-30.		H0437: 2, L0769: 2, S0028: 2, L0439: 2, S0436: 2, H0556: 1, H0125: 1, S0420: 1, H0619: 1, H0587: 1, H0635: 1, H0253: 1, H0318: 1, H0744: 1, H0052: 1, H0009: 1, H0172: 1, H0266: 1,	1		

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90	HWLBO67	834315	100	42 - 161	207				S0374: 1	2		
91	HWLGP26	834770	101	1091 - 1306	208			Pro-26 to Met-35.	L0766: 5, L0803: 5, L0794: 3, S0410: 2, H0551: 2, H0435: 2, L0756: 2, L0731: 2, H0585: 1, S0212: 1, S0282: 1, L0534: 1, S0442: 1, S0354: 1, H0735: 1, H0486: 1, H0014: 1, H0354: 1, H0644: 1, H0135: 1, H0647: 1, L0369: 1, L0640: 1, L0763: 1, L0770: 1, L3905: 1, L0646: 1, L0771: 1, L0804: 1, L0784: 1, L0528: 1, L0789: 1, L0790: 1, L0792: 1, L3827: 1, H0658: 1, L0749: 1, L0758: 1 and S0436: 1.			
92	HILCA24	869856	102	191 - 1174	209			Gln-52 to Arg-57, Glu-74 to Leu-84, Val-104 to Asp-110, Gly-157 to Gly-163,	L0748: 4, H0090: 2, L0659: 2, H0521: 2, L0777: 2, L0608: 2, H0543: 2, T0002: 1,			

						Asn-185 to Ser-195, Arg-245 to Asp-250, Pro-302 to Pro-310, Thr-316 to Tyr-322.	S0114: 1, L3658: 1, S0358: 1, S0408: 1, L3649: 1, T0109: 1, H0581: 1, H0622: 1, H0031: 1, H0644: 1, S0002: 1, L0657: 1, L0526: 1, L0789: 1, L0664: 1, S0380: 1, H0522: 1, L0749: 1 and L0779: 1.		
	HILCA24	782450	110	189 - 1172	217	Gln-52 to Arg-57, Glu-74 to Leu-84, Val-104 to Asp-110, Gly-157 to Gly-163, Asn-185 to Ser-195, Arg-245 to Asp-250, Pro-302 to Pro-310, Thr-316 to Tyr-322.			
93	HE2CA60	888705	103	1731 - 1754	210		H0305: 16, L0777: 11, L0471: 10, S0422: 9, L0766: 9, H0624: 8, H0013: 7, H0170: 6, L2551: 6, H0046: 6, L0665: 6, L0598: 5, L0662: 5, L0776: 5, H0547: 5, L0758: 5, L0589: 5, H0171: 4, L0659: 4, L0666: 4, L0663: 4, L0756: 4, L0731: 4, S0358: 3, L2744: 3, L3655: 3,	17	

						H0581: 3, H0457: 3, S0406: 3, L0744: 3, L0439: 3, L0752: 3, S0436: 3, H0542: 3, H0543: 3, L3643: 2, H0650: 2, H0657: 2, S0116: 2, S0442: 2, S0354: 2, L0717: 2, S0414: 2, H0486: 2, T0040: 2, H0318: 2, H0421: 2, H0428: 2, H0553: 2, H0090: 2, H0040: 2, H0063: 2, H0641: 2, L0769: 2, L0761: 2, L0764: 2, L0650: 2, L0774: 2, L0805: 2, L0657: 2, H0144: 2, L3811: 2, L3832: 2, H0521: 2, S0404: 2, L0741: 2, L0740: 2, L0747: 2, L0759: 2, S0434: 2, L0362: 2, H0685: 1, S0218: 1, L0785: 1, H0341: 1, H0255: 1, H0663: 1, H0662: 1, H0402: 1, S0376: 1, S0360: 1, S0410: 1, L3645: 1, L3646: 1, H0637: 1, H0741: 1, H0722: 1, H0735: 1,	
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	HE2CA60	770301	111	360 - 383	218						
94	HPWTF23	844775	104	283 - 675	211	Val-80 to Leu-92, Ser-98 to Lys-104, Pro-111 to Pro-122.		S0474: 47, H0710: 28, L0747: 18, L0659: 17, H0656: 13, H0436: 10, H0271: 9, L0751: 9, H0581: 8, H0179: 8, H0063: 8, L0731: 8, L0599: 8, H0740: 7, L0756: 7, H0650: 6, L0662: 6, H0555: 6, S0354: 5, H0728: 5, H0733: 5, H0734: 5, H0036: 5, H0590: 5, H0052: 5, L0770: 5, S0428: 5, S0374: 5, L0439: 5, L3643: 4, H0717: 4, H0747: 4, H0393: 4, S0222: 4, H0156: 4, H0309: 4, S0312: 4, S0314: 4, H0090: 4, H0591: 4,			

								L0752: 1, L0755: 1, H0707: 1, L0591: 1, L0592: 1, H0653: 1, H0136: 1, S0412: 1 and H0721: 1.			
	HPWTF23	843700	112	283 - 675	219	Val-80 to Leu-92, Ser-98 to Lys-104, Pro-111 to Pro-122.					
95	HGCAC19	851527	105	317 - 346	212			L0794: 15, L0803: 12, L0766: 7, H0013: 6, H0090: 6, L0663: 6, L0777: 6, L0731: 6, L0759: 6, H0457: 5, H0328: 5, L0493: 5, L0666: 5, L0754: 5, L0749: 5, H0543: 5, H0656: 4, S0358: 4, H0615: 4, L0665: 4, H0521: 4, L0779: 4, L0588: 4, H0305: 3, S0360: 3, H0036: 3, H0052: 3, T0042: 3, L0761: 3, L0805: 3, L0809: 3, H0144: 3, H0670: 3, H0696: 3, L0591: 3, S0134: 2, H0657: 2, L3659: 2, S0418: 2, S0442: 2, S0007: 2, S0045: 2, L0717: 2, H0600: 2, H0486: 2, H0156: 2,			

					S0474: 1, H0263: 1, T0115: 1, H0545: 1, L0157: 1, H0123: 1, L0471: 1, H0015: 1, S0388: 1, S0051: 1, H0375: 1, H0271: 1, H0188: 1, S0312: 1, S0003: 1, H0688: 1, H0039: 1, H0622: 1, H0031: 1, H0644: 1, L0055: 1, H0169: 1, L0456: 1, H0163: 1, H0634: 1, H0551: 1, H0379: 1, H0488: 1, H0279: 1, L0475: 1, S0352: 1, H0652: 1, S0208: 1, L0640: 1, L0763: 1, L0500: 1, L0769: 1, L0646: 1, L0662: 1, L0649: 1, L0498: 1, L0804: 1, L0650: 1, L0784: 1, L0806: 1, L0653: 1, L0606: 1, L0515: 1, L0659: 1, L0526: 1, L0519: 1, L0788: 1, L0790: 1, L0791: 1, L0664: 1, S0053: 1, S0296: 1, H0547: 1, S0126: 1, H0682: 1, H0684: 1, H0658: 1,	
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	HGCAC19	842540	113	315 - 344	220							
	HGCAC19	801999	114	317 - 346	221							
96	HEQBJ01	876546	106	2603 - 2662	213			S0360: 3, H0619: 3, H0673: 2, L0438: 2, H0685: 1, S0444: 1, H0544: 1, H0266: 1, H0163: 1, L0770: 1, L0646: 1, L0768: 1, L0766: 1, L0803: 1, L0776: 1, S0152: 1, S0027: 1, L0439: 1, L0747: 1, L0777: 1, L0752: 1 and L0758: 1.	16			
	HEQBJ01	861786	115	2603 - 2662	222							
	HEQBJ01	834633	116	505 - 564	223							
97	HBJHT01	587262	107	200 - 265	214			L0667: 2, S0114: 1, H0351: 1, H0318: 1, H0615: 1 and L0764: 1.				
	HBJHT01	580026	117	193 - 336	224							
98	HAGDW20	637489	108	238 - 291	215			S0010: 1 and H0616: 1.				

99	HTLJF11	843506	109	933 - 1049	216	Pro-4 to Gly-9.	H0253: 7, H0618: 4, H0620: 3, L0794: 3, L0769: 2, L0768: 2, L0439: 2, H0327: 1, H0051: 1, S0250: 1, S0036: 1, L0639: 1, L0761: 1, L0635: 1, L0791: 1, L0664: 1, L0438: 1, H0539: 1, L0741: 1, L0747: 1, L0750: 1, L0756: 1 and L0753: 1.		
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Table 1B.2

Gene No:		cDNA Clone ID	Contig ID:	SEQ ID NO:X	Tissue Distribution Library Code:Count (see Table 4 for Library Codes)			
1		HACBT91	789939	11	AR283:41, AR219:39, AR277:38, AR218:33, AR055:29, AR316:28, AR039:25, AR104:22, AR299:22, AR096:20, AR089:20, AR185:19, AR240:19, AR282:18, AR060:17, AR300:16, AR313:16 L0665:5, L0743:3, H0341:2, L0761:2, L0756:2, S0356:1, H0734:1, S0280:1, T0048:1, H0271:1, S0440:1, H0641:1, H0646:1, L0770:1, L0637:1, L0800:1, L0773:1, L0648:1, L0662:1, L0768:1, L0766:1, L0649:1, L0375:1, L0784:1, L0806:1, L0655:1, L0809:1, H0672:1, S0406:1, L0747:1, L0749:1 and L0750:1.			
2		HADDE71	839187	12	AR283:48, AR277:39, AR313:33, AR219:30, AR316:27, AR282:27, AR089:27, AR299:26, AR218:26, AR240:25, AR185:22, AR104:22, AR055:22, AR096:22, AR300:20, AR039:20, AR060:15 L0769:11, L0747:9, L0809:6, S0408:4, L0770:4, L0439:4, L0752:4, L0759:4, L0766:3, L0803:3, L0666:3, L0751:3, L0780:3, S0007:2, H0619:2, H0351:2, H0333:2, H0427:2, H0052:2, L0761:2, L0662:2, L0794:2, L0774:2, L0806:2, L0659:2, H0547:2, H0521:2, L0741:2, L0745:2, L0750:2, L0779:2, L0777:2, H0543:2, H0739:1, H0171:1, L3019:1, H0483:1, H0254:1, H0125:1, H0675:1, H0580:1, H0722:1, H0733:1, S0140:1, H0261:1, H0592:1, H0586:1, H0587:1, H0257:1, H0486:1, L0022:1, H0042:1, H0581:1, H0150:1, H0086:1, H0123:1, T0010:1, H0266:1, H0673:1, S0364:1, H0087:1, H0264:1, H0494:1, H0560:1, H0538:1, L0762:1, L0772:1, L0646:1, L0765:1, L0649:1, L0805:1, L0776:1, L0657:1, L0783:1, L5622:1, L0791:1, L2654:1, S0126:1, H0435:1, S0330:1, H0522:1, L0750:1.			

3	HADDJ13	827273			L0743:1, L0744:1, L0749:1, L0786:1, L0753:1, L0755:1, L0731:1, L0758:1, S0436:1, S0011:1 and S0192:1.
4	HADMA77	783049	13	H0427:1	
			14		AR104:16, AR039:10, AR277:9, AR089:9, AR240:9, AR055:9, AR300:8, AR218:7, AR299:7, AR283:7, AR060:7, AR282:7, AR316:6, AR219:6, AR096:5, AR185:5, AR313:4, L0439:15, S0222:4, L0157:4, L0769:4, L0438:3, L0745:3, L0731:3, L0758:3, L0599:3, H0443:2, H0441:2, S0010:2, L0662:2, L0744:2, L0748:2, L0750:2, L0756:2, L0777:2, H0583:1, L0005:1, S0354:1, H0675:1, S0408:1, H0619:1, H0369:1, H0574:1, H0486:1, H0390:1, S0346:1, H0309:1, H0597:1, T0003:1, H0024:1, S0628:1, H0028:1, T0006:1, H0628:1, H0135:1, H0551:1, S0438:1, L0520:1, L0768:1, L0776:1, L0559:1, L0659:1, L0384:1, L0809:1, H0144:1, H0547:1, L0746:1, L0747:1, L0757:1 and S0434:1.
5	HADMB15	847116	15		AR104:19, AR218:19, AR219:16, AR089:11, AR313:8, AR055:8, AR060:7, AR299:6, AR282:5, AR300:5, AR039:5, AR240:5, AR316:5, AR185:5, AR277:4, AR283:4, AR096:3, L0595:2, L0442:1, L0005:1, L3653:1, H0390:1, H0081:1, H0024:1, L0770:1, L5566:1, L0651:1, L0565:1, L0439:1, L0747:1, L0752:1, H0445:1, L0592:1 and L0599:1.
6	HAGBQ12	722205	16		AR060:7, AR055:6, AR104:5, AR185:5, AR089:4, AR299:4, AR277:4, AR300:4, AR283:4, AR313:3, AR316:3, AR240:3, AR039:3, AR096:3, AR282:2, AR218:2, AR219:1, L0754:4, L0805:2, L0777:2, L0755:2, S0010:1, H0049:1, L0163:1, L0771:1, L0775:1 and L0776:1.
7	HAGCC87	638587	17		AR313:17, AR039:14, AR277:10, AR104:10, AR089:9, AR300:9, AR096:9, AR299:8, AR185:7, AR055:6, AR060:6, AR218:6, AR240:6, AR316:6, AR282:4, AR283:4, AR219:3, L0439:4, L0519:3, S0010:2, T0010:1, L0809:1, H0682:1, S0404:1, S0406:1, H0436:1 and L0756:1.
8	HAGHN57	773286	18		AR313:12, AR316:11, AR218:11, AR185:11, AR039:10, AR219:10, AR299:10, AR060:9, AR055:8, AR277:8, AR282:8, AR096:7, AR089:7, AR300:7, AR240:6, AR104:6, AR283:4, H0521:5, L0777:5, S0376:4, H0733:3, H0156:3, H0519:3, H0436:3, L0731:3, H0656:2, H0580:2, H0747:2, L3816:2, H0036:2, L0471:2, H0090:2, H0040:2, H0551:2, H0494:2, S0438:2, S0440:2, H0529:2, L0809:2, H0144:2, S0374:2, H0593:2, H0170:1, L3643:1, H0583:1, H0650:1, S0418:1, S0358:1, S0444:1, L3645:1, H0741:1, H0734:1, S0045:1, S0476:1, H0619:1, H0586:1, H0643:1, H0632:1, H0486:1, S0280:1, H0590:1, S0010:1, S0346:1, H0581:1, H0231:1, S6028:1, H0687:1, S0003:1, S0214:1, H0252:1, H0615:1, H0212:1, L0455:1, S0366:1, H0163:1, H0038:1, T0067:1, L0475:1, H0560:1, H0561:1, S0464:1, H0646:1, S0426:1, H0026:1, L0790:1, H0520:1, H0435:1, S0328:1, H0539:1, H0704:1, S0027:1, L0439:1, L0750:1, L0756:1, L0757:1, S0434:1, L0581:1, L0595:1, H0543:1 and H0423:1.
9	HAGHR18	655435	19		AR052:6, AR055:6, AR247:6, AR061:6, AR053:6, AR060:5, AR182:5, AR263:5, AR310:5, AR312:4, AR251:4, AR033:4, AR244:4, AR293:4, AR282:4, AR269:3, AR185:3, AR270:3, AR298:3, AR089:3, AR253:3, AR296:3, AR104:3, AR232:3, AR299:3, AR285:3, AR198:3, AR286:3, AR184:3, AR237:3, AR277:3, AR295:3, AR300:3, AR213:3, AR284:2, AR283:2, AR267:2, AR266:2, AR290:2, AR268:2, AR289:2, AR313:2, AR316:2, AR294:2, AR186:2, AR096:2, AR183:2, AR233:2, AR240:2, AR229:2, AR218:2, AR177:2, AR259:2, AR246:2, AR248:2, AR175:2, AR292:2, AR309:2, AR265:2, AR226:1, AR039:1, AR234:1, AR179:1, AR258:1, AR219:1, AR231:1, AR238:1, L0717:1 and S0346:1.
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17	HBNAX40	834801	27	AR218:8, AR313:8, AR055:6, AR060:6, AR089:5, AR104:5, AR185:4, AR300:4, AR299:4, AR316:4, AR096:3, AR277:3, AR240:3, AR039:3, AR283:3, AR282:3, AR219:2 L0439:11, H0171:5, L0754:5, L0748:4, H0052:3, L0662:3, L0756:3, L0755:3, H0422:3, S0360:2, L0738:2, H0032:2, L0803:2, L0655:2, L0789:2, L0605:2, H0423:2, H0638:1, T0114:1, H0156:1, L0021:1, S0010:1, H0581:1, H0046:1, L0471:1, H0014:1, H0356:1, H0188:1, H0553:1, H0591:1, S0386:1, T0042:1, H0625:1, H0641:1, S0142:1, L0598:1, L0369:1, L0640:1, L0375:1, L0654:1, L0659:1, L0783:1, L0663:1, L0665:1, H0144:1, L0352:1, H0547:1, H0648:1, H0672:1, H0555:1, H0436:1, L0749:1, L0779:1, L0731:1, L0758:1, L0759:1, H0445:1, L0366:1 and H0668:1.
18	HBXCX15	637542	28	S0038:3, H0438:1, L0363:1 and S0053:1.
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21	HCEZF82	831745	31		L0748:1, H0052:8, L0803:8, L0749:8, L0770:7, L0439:5, L0746:4, L0752:4, L3811:3, H0575:2, H0012:2, H0031:2, L0768:2, L0804:2, L0774:2, L0740:2, L0747:2, L0756:2, L0779:2, L0757:2, L0758:2, L0592:2, L0593:2, H0556:1, S0420:1, S0376:1, H0441:1, H0632:1, S0010:1, T0115:1, H0545:1, H0009:1, H0620:1, H0197:1, H0051:1, S0388:1, S0051:1, H0252:1, H0032:1, L0455:1, H0591:1, H0272:1, L0564:1, S0438:1, S0344:1, L0373:1, L0646:1, L0794:1, L0766:1, L0805:1, L0776:1, L0783:1, L0809:1, S0374:1, H0522:1, H0134:1, L0780:1, L0731:1, L0759:1, S0436:1, L0597:1, H0543:1, H0423:1 and L0600:1.
22	HCUCF89	637986	32		AR313:26, AR039:18, AR277:13, AR299:12, AR096:11, AR089:11, AR185:11, AR300:10, AR240:8, AR316:8, AR218:5, AR282:4, AR104:4, AR060:4, AR219:3, AR055:2 H0306:1, L0761:1 and H0436:1.
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27	HDPIE44	899328	37		AR263:6, AR265:3, AR184:3, AR183:3, AR096:3, AR313:3, AR269:3, AR039:3, AR104:2, AR312:2, AR270:2, AR268:2, AR298:2, AR296:2, AR292:2, AR060:2, AR052:2, AR282:2, AR291:2, AR198:2, AR316:2, AR192:2, AR299:2, AR286:2, AR267:2, AR218:2, AR055:2, AR295:2, AR290:2, AR283:2, AR089:2, AR289:1, AR231:1, AR213:1, AR247:1, AR284:1, AR293:1, AR053:1, AR033:1, AR238:1, AR258:1, AR182:1, AR177:1, AR277:1, AR185:1, AR310:1 L3811:7, L0439:7, L0759:5, L0591:5, L0803:4, H0547:4, L0748:4, L0755:4, L0596:4, H0171:3, S0376:3, S0007:3, H0024:3, H0355:3, H0615:3, H0428:3, H0090:3, H0623:3, S0422:3, L0794:3, L0766:3, L0659:3, H0144:3, H0658:3, S0406:3, L0749:3, L0758:3, S0436:3, H0624:2, H0717:2, S0358:2, S0360:2, H0486:2, H0427:2, S0010:2, H0052:2, H0251:2, H0687:2, H0622:2, H0553:2, H0644:2, H0591:2, S0438:2, L0769:2, L0662:2, L0805:2, S0374:2, S0126:2, H0689:2, H0670:2, H0521:2, S0028:2, L0744:2, L0740:2, L0754:2, L0752:2, L0593:2, S0192:2, H0506:2, H0265:1, H0294:1, H0656:1, S0212:1, L0481:1, S0418:1, L0005:1, S0356:1, S0442:1, S0408:1, H0733:1, H0208:1, S0045:1, H0619:1, L0717:1, S0222:1, H0455:1, L3653:1, H0013:1, H0599:1, S0474:1, H0196:1, H0263:1, H0046:1, H0172:1, H0050:1, L0471:1, H0012:1, H0620:1, H0014:1, H0051:1, H0356:1, H0375:1, S0316:1, H0328:1, H0688:1, L0483:1, S0364:1, S0366:1, H0135:1, H0163:1, H0038:1, H0040:1, H0634:1, H0551:1, H0488:1, T0042:1, H0494:1, S0016:1, H0625:1, H0561:1, S0440:1, L2270:1, S0344:1, L3818:1, H0538:1, L0598:1, L0770:1, L0638:1, L0641:1, L0626:1, L0804:1, L0375:1, L0784:1, L0523:1, L0806:1, L0776:1, L0526:1, L0809:1, L5622:1, L0789:1, L0793:1, L4559:1, L0663:1, L4560:1, L3826:1, L3828:1, H0683:1, H0672:1, H0651:1, S0330:1, H0539:1, H0555:1, S0390:1, S0206:1, L0747:1, L0779:1, S0308:1, L0604:1 and H0423:1.
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30	HDTLM18	836057	40	AR202:68, AR194:68, AR281:64, AR244:59, AR315:56, AR205:52, AR246:50, AR280:49, AR283:45, AR314:39, AR271:38, AR232:37, AR243:37, AR241:35, AR316:34, AR282:33, AR204:33, AR263:32, AR089:32, AR192:32, AR265:31, AR277:31, AR206:30, AR219:29, AR310:29, AR033:29, AR096:29, AR313:28, AR299:28, AR240:26, AR247:26, AR273:24, AR300:24, AR198:24, AR295:24, AR274:24, AR218:24, AR039:23, AR275:23, AR055:23, AR213:23, AR104:22, AR251:22, AR238:20, AR177:20, AR312:20, AR060:19, AR226:19, AR052:19, AR231:18, AR053:18, AR309:18, AR234:18, AR227:18, AR185:17, AR292:17, AR237:17, AR229:16, AR258:16, AR183:16, AR175:15, AR294:14, AR256:13, AR259:13, AR233:13, AR293:11, AR186:11, AR253:10, AR061:10, AR266:10, AR267:9, AR285:8, AR248:8, AR270:8, AR296:8, AR284:7, AR179:7, AR289:7, AR249:7, AR268:6, AR269:6, AR291:6, AR184:6, AR298:5, AR286:5, AR182:5, AR290:4, L0794:6, L0748:6, H0556:5, L0771:5, H0052:4, L0756:4, L0596:4, H0265:3, H0341:3, H0587:3, L0662:3, L0803:3, L0790:3, S0152:3, L0750:3, S0114:2, S0360:2, H0318:2, L0471:2, L0369:2, L0763:2, L0770:2, L0764:2, L0766:2, L0774:2, L0378:2, L0789:2, L0666:2, L3825:2, H0547:2, L0747:2, L0777:2, L0581:2, H0543:2, H0422:2, S0218:1, H0255:1, S0418:1, S0354:1, S0376:1, S0408:1, L3649:1, S0045:1, H0747:1, H0619:1, L0717:1, S0222:1, H0431:1, H0586:1, H0013:1, H0069:1, S0049:1, H0009:1, H0071:1, H0083:1, H0428:1, T0006:1, H0424:1, H0213:1, H0644:1, H0628:1, H0135:1, H0163:1, H0616:1, H0413:1, H0059:1, H0561:1, S0448:1, H0647:1, L3818:1, S0002:1, L0769:1, L0800:1, L0363:1, L0767:1, L0768:1, L0649:1, L0804:1, L0806:1, L0657:1, L0512:1, L0659:1, L0384:1, L0647:1, L5622:1, L5623:1, L0664:1, L0665:1, S0374:1, L3828:1, S0126:1, H0711:1, H0658:1, H0666:1, H0539:1, H0753:1, H0521:1, H0522:1, S0406:1, H0555:1, H0436:1, L0439:1, L0749:1, S0031:1, L0595:1, H0136:1, H0542:1, H0423:1, S0424:1 and H0352:1.
31	HE6CS65	762960	41	AR313:9, AR089:5, AR299:5, AR185:4, AR300:3, AR060:3, AR096:3, AR282:3, AR039:2, AR316:2, AR055:2, AR104:2, AR240:1, AR277:1, AR283:1, H0486:1 and L0599:1. AR219:61, AR277:59, AR218:48, AR283:47, AR282:43, AR316:39, AR089:38, AR313:36, AR299:34, AR240:33, AR104:29, AR055:29, AR096:29, AR039:27, AR185:26, AR300:22, AR060:21, L0777:16, L0748:12, L0757:11, L0776:8, L0439:7, H0692:6, H0046:6, L0769:5, L0666:5, S0242:5, L0770:4, L0771:4, L0438:4, L0743:4, L0754:4, L0749:4, L0758:4, S0444:3, H0051:3, L0662:3, L0766:3, S0378:3, L0751:3, L0747:3, S0436:3, S0212:2, H0637:2, H0497:2, H0545:2, H0050:2, H0031:2, H0090:2, H0100:2, L0768:2, L0561:2, L0774:2, L0775:2, L0657:2, H0670:2, S3014:2, L0744:2, L0752:2, L0581:2, H0624:1, H0170:1, H0713:1, H0717:1, S6024:1, T0049:1, H0255:1, S0356:1, S0442:1, S0358:1, S0376:1, S0360:1, H0619:1, L3651:1, L0717:1, S0278:1, H0391:1, H0333:1, H0013:1, H0053:1, H0575:1, S0346:1, H0052:1, H0263:1, H0596:1, L0738:1, H0572:1, H0510:1, H0266:1, H0688:1, H0039:1, H0622:1, H0111:1, H0181:1, H0617:1, H0032:1, H0169:1, H0634:1, H0087:1,

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35	HEBFR46	847064	45	AR313:58, AR039:47, AR300:30, AR096:29, AR299:29, AR277:28, AR089:27, AR185:27, AR316:22, AR219:22, AR104:21, AR218:20, AR240:20, AR282:15, AR060:15, AR055:11, AR283:7 H0457:10, H0550:5, H0436:5, H0549:4, H0616:4, L0519:4, H0556:3, H0580:3, S0007:3, S0046:3, L0809:3, L0747:3, L0777:3, S0436:3, H0295:2, T0040:2, H0266:2, L0761:2, L0783:2, L0789:2, H0658:2, H0521:2, L0753:2, L0731:2, L0596:2, H0543:2, S0040:1, S0116:1, S0282:1, H0662:1, H0402:1, H0125:1, L0534:1, L0562:1, S0356:1, S0358:1, H0749:1, L3816:1, H0559:1, H0069:1, H0599:1, H0618:1, H0253:1, H0581:1, H0546:1, H0123:1, S0051:1, H0083:1, H0687:1, H0284:1, H0124:1, H0038:1, H0551:1, H0623:1, S0038:1, T0041:1, S0440:1, S0150:1, L3818:1, S0002:1, L0763:1, L0769:1, L5575:1, L0627:1, L0800:1, L0662:1, L0803:1, L0793:1, L0666:1, L2264:1, L3825:1, L3827:1, L3828:1, H0547:1, H0519:1, H0539:1, S0037:1, S0206:1, L0748:1, L0749:1, H0595:1, L0593:1, S0194:1 and S0276:1.
36	HEOMC46	866171	46	AR277:48, AR283:34, AR219:31, AR218:30, AR316:28, AR313:27, AR282:27, AR089:25, AR299:23, AR240:23, AR096:22, AR039:22, AR104:20, AR185:20, AR300:19, AR055:19, AR060:13 H0749:2, H0581:2, H0457:2 and S0116:1.
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42	HGBGN34	648659	52		AR240:13, AR060:11, AR055:10, AR218:7, AR096:7, AR185:7, AR282:7, AR089:6, AR283:6, AR300:6, AR299:6, AR104:5, AR316:5, AR277:4, AR313:3, AR039:3, AR219:2, L0747:5, H0716:2, H0427:2, S0280:2, H0662:1, S0444:1, H0441:1, H0492:1, T0001:1, H0014:1, H0030:1, H0674:1, L5575:1, L0659:1, S0330:1, L0752:1 and S0436:1.
43	HGLBG15	701990	53		AR055:14, AR162:7, AR161:7, AR163:7, AR104:6, AR060:6, AR218:6, AR191:6, AR089:6, AR197:6, AR261:6, AR269:5, AR170:5, AR176:5, AR165:5, AR185:5, AR188:5, AR164:5, AR189:5, AR291:5, AR166:5, AR246:5, AR190:5, AR096:5, AR257:4, AR240:4, AR243:4, AR288:4, AR309:4, AR272:4, AR316:4, AR275:4, AR270:4, AR175:4, AR290:4, AR274:4, AR053:4, AR219:4, AR289:4, AR264:4, AR255:4, AR199:4, AR268:4, AR204:4, AR286:4, AR271:4, AR039:4, AR205:3, AR173:3, AR196:3, AR233:3, AR287:3, AR262:3, AR215:3, AR267:3, AR300:3, AR200:3, AR177:3, AR296:3, AR181:3, AR283:3, AR299:3, AR295:3, AR239:3, AR311:3, AR282:3, AR297:3, AR174:3, AR285:3, AR294:3, AR293:3, AR168:3, AR201:3, AR238:3, AR235:3, AR217:3, AR313:3, AR198:3, AR312:3, AR258:3, AR203:3, AR178:3, AR254:3, AR211:2, AR263:2, AR247:2, AR193:2, AR237:2, AR266:2, AR277:2, AR256:2, AR182:2, AR171:2, AR210:2, AR250:2, AR260:2, AR195:2, AR172:2, AR207:2, AR236:2, AR232:2, AR221:2, AR226:2, AR224:2, AR216:1, AR179:1, AR033:1, AR061:1, AR252:1, L0803:19, S0474:17, L0748:13, S0408:11, H0351:11, L2669:11, L2504:10, L0770:10, L0805:9, L0439:9, L0754:9, S0422:8, L0809:8, L0794:6, L0755:6, L0731:6, L0758:6, S0360:5, H0265:4, S0414:4, H0581:4, H0271:4, L0771:4, L0804:4, L0776:4, L0659:4, L0666:4, L0749:4, L0591:4, H0327:3, L0806:3, L0655:3, L0636:3, L0565:3, H0436:3, L0777:3, S0434:3, S0436:3, S0412:3, S0116:2, S0212:2, H0661:2, S0358:2, S0132:2, L3388:2, S0222:2, H0123:2, H0266:2, S0003:2, H0031:2, H0551:2, L0598:2, L0638:2, L0662:2, L0766:2, L0650:2, L0664:2, L0665:2, S0374:2, H0547:2, H0435:2, H0660:2, S0378:2, L0740:2, L0750:2, L0756:2, L0752:2, H0624:1, H0556:1, S0040:1, H0295:1, S0114:1, H0656:1, L2904:1, S0001:1, H0671:1, S0356:1, S0442:1, S0376:1, S0444:1, H0675:1, H0730:1, H0741:1, H0208:1, S0045:1, S0476:1, H0393:1, H0550:1, H0431:1, H0586:1, H0642:1, L3499:1, H0013:1, H0069:1, H0635:1, H0427:1, H0156:1, L0021:1, H0042:1, T0082:1, H0590:1, S0010:1, H0318:1, H0251:1, H0596:1, L0040:1, H0545:1, H0457:1, H0009:1, N0006:1, L0471:1, H0024:1, H0051:1, H0083:1, H0061:1, S0316:1, H0687:1, H0688:1, H0644:1, H0617:1, H0591:1, H0038:1, H0040:1, H0616:1, H0264:1,

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45	HHSDI53	862028	55	<p>AR313:45, AR039:43, AR300:22, AR299:22, AR096:21, AR316:20, AR185:19, AR089:19, AR277:19, AR219:15, AR240:14, AR104:14, AR218:13, AR282:12, AR060:11, AR055:8, AR283:4, L0766:10, L0752:8, L0439:6, L0747:6, L0740:5, L0756:5, S0408:4, L0779:4, L0777:4, L0731:4, S0051:3, H0169:3, L0803:3, L0774:3, L0809:3, L0754:3, S0360:2, H0574:2, S0422:2, L0763:2, L0805:2, L0666:2, L0663:2, L0751:2, L0755:2, L0759:2, L0601:2, H0624:1, S0040:1, H0713:1, S0114:1, S0298:1, S0420:1, S0444:1, H0580:1, H0730:1, H0733:1, L3388:1, H0351:1, H0600:1, H0331:1, H0013:1, L0021:1, H0575:1, H0590:1, T0110:1, H0012:1, H0615:1, H0031:1, H0553:1, S0440:1, H0646:1, S0002:1, L0772:1, L0645:1, L0773:1, L0662:1, L0794:1, L0381:1, L0775:1, L0776:1, L0657:1, L0659:1, L5622:1, L0790:1, H0547:1, H0648:1, H0539:1, S0152:1, H0696:1, S0044:1, S0406:1, S0028:1, L0758:1, S0434:1, S0436:1, L0366:1, S0011:1, S0276:1, H0422:1, S0398:1 and S0424:1.</p>
46	HISBA38	561711	56	AR277:1, L0766:3, H0318:1 and H0539:1.
47	HJPBK28	638191	57	<p>L0794:6, L0439:5, L0759:5, H0556:4, L0771:4, L0770:3, L0643:3, H0144:3, H0156:2, H0188:2, H0090:2, H0641:2, L0662:2, L0766:2, L0803:2, L0776:2, L0661:2, L0659:2, L0790:2, H0522:2, S0436:2, H0295:1, T0049:1, H0583:1, S0116:1, H0663:1, H0662:1, S0356:1, S0376:1, S0132:1, H0586:1, H0587:1, H0486:1, H0575:1, H0748:1, H0744:1, H0309:1, H0231:1, H0083:1, H0271:1, H0286:1, H0622:1, H0031:1, L0455:1, H0068:1, H0063:1, H0551:1, H0264:1, H0268:1, T0041:1, H0494:1, H0633:1, L0637:1, L3905:1, L0800:1, L0775:1, L0806:1, L0383:1, L0809:1, L0666:1, L0663:1, L0664:1, L2264:1, L3827:1, L3828:1, H0519:1, H0593:1, H0435:1, H0672:1, H0436:1, S0027:1, L0740:1, L0749:1, L0731:1, L0757:1,</p>

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49	HLYGE16	651339	59	AR313:6, AR316:5, AR218:3, AR300:3, AR299:3, AR055:3, AR185:2, AR039:2, AR096:2, AR277:2, AR219:1, AR089:1 H0692:10, L0777:10, L0805:5, L0803:3, L2497:2, H0328:2, L0662:2, L0794:2, L0809:2, L3832:2, L0748:2, L0752:2, L0599:2, H0170:1, H0402:1, S0444:1, S0360:1, H0747:1, L2486:1, L3503:1, H0427:1, H0644:1, H0038:1, L0800:1, L0648:1, L0804:1, H0670:1, H0478:1, L0731:1, L0758:1, H0445:1, S0434:1, L0591:1 and L0362:1.
50	HLYGY91	658703	60	AR104:113, AR219:90, AR218:87, AR089:82, AR283:79, AR277:79, AR313:78, AR055:75, AR240:71, AR316:70, AR185:63, AR282:60, AR299:59, AR096:54, AR039:50, AR060:48, AR300:38 L0659:10, T0040:9, L0665:9, L0759:9, L0519:8, L0776:7, S0436:7, L0744:6, L0747:6, L0749:6, L0758:6, S0418:5, H0052:5, H0457:5, H0150:5, L0769:5, L0766:5, L0748:5, H0265:4, S0420:4, S0356:4, S0360:4, S0046:4, S0010:4, H0545:4, H0687:4, H0494:4, S0440:4, L0662:4, L0768:4, L0774:4, L0775:4, L0751:4, L0754:4, L0779:4, H0484:3, H0734:3, H0549:3, H0599:3, H0421:3, H0620:3, S0051:3, L0764:3, L0666:3, H0435:3, H0648:3, H0539:3, L0596:3, H0543:3, H0624:2, H0171:2, H0556:2, H0295:2, H0657:2, H0656:2, S0354:2, S0358:2, S0376:2, S0408:2, S0007:2, S0132:2, S0476:2, S0222:2, H0486:2, T0039:2, H0635:2, H0156:2, H0618:2, T0048:2, H0581:2, H0544:2, H0373:2, H0428:2, T0006:2, H0604:2, H0031:2, H0551:2, T0067:2, H0264:2, H0647:2, S0344:2, L0638:2, L0372:2, L0641:2, L0806:2, L0653:2, L0527:2, L0809:2, L0565:2, L0438:2, H0519:2, H0689:2, H0658:2, H0672:2, S0330:2, S0406:2, H0436:2, S0027:2, L0750:2, S0434:2, L0605:2, S0194:2, H0506:2, H0685:1, H0713:1, H0717:1, H0740:1, H0294:1, S0212:1, S0110:1, S0282:1, H0483:1, S0442:1, H0637:1, H0733:1, S0468:1, H0747:1, L3388:1, H0351:1, H0550:1, H0587:1, H0642:1, H0559:1, L0622:1, L3653:1, H0013:1, H0250:1, H0069:1, S0280:1, H0706:1, S0346:1, H0705:1, H0318:1, S0049:1, H0748:1, L0040:1, H0597:1, L0738:1, H0009:1, H0563:1, H0123:1, H0050:1, L0471:1, H0012:1, H0024:1, H0014:1, S0388:1, H0239:1, H0594:1, S0628:1, H0271:1, H0292:1, H0213:1, H0628:1, H0673:1, H0068:1, S0036:1, H0135:1, H0090:1, H0038:1, H0634:1, H0087:1, H0488:1, H0268:1, H0412:1, H0413:1, S0038:1, T0042:1, H0560:1, H0641:1, S0210:1, S0422:1, S0002:1, H0529:1, L0770:1, L0637:1, L3905:1, L5566:1, L0761:1, L0772:1, L0646:1, L0374:1, L0771:1, L4500:1, L0651:1, L0784:1, L0807:1, L0657:1, L0658:1, L0656:1, L0782:1, L0783:1, L0530:1, L0647:1, L0788:1, L0663:1, L0664:1, S0216:1, H0693:1, L3826:1, H0520:1, H0547:1, S0126:1, H0682:1, H0659:1, S0328:1, S0380:1, H0710:1, H0521:1, H0522:1, H0627:1, S0028:1, L0741:1, L0742:1, L0439:1, L0740:1, L0756:1, L0786:1, L0780:1, L0755:1, L0581:1, L0595:1, L0601:1, H0667:1, S0192:1, H0542:1, L0718:1 and S0424:1.
52	HMIK10	562774	62	AR055:7, AR218:7, AR060:6, AR219:6, AR185:4, AR283:4, AR240:4, AR300:4, AR104:3, AR089:3, AR299:3, AR039:3, AR316:2, AR277:2, AR096:2, AR313:2, AR282:2 S0628:1

53	HMBD93	634227	63	AR277:111, AR283:78, AR219:72, AR316:65, AR104:61, AR055:58, AR089:55, AR218:54, AR313:49, AR282:48, AR299:47, AR039:44, AR185:41, AR096:39, AR240:36, AR060:33, AR300:31 L0439:6, L0751:5, L0770:3, L0769:3, L0764:3, H0617:2, L0766:2, L0752:2, H0445:2, S0624:1, H0351:1, S0222:1, H0586:1, S0010:1, S0628:1, L0768:1, L0794:1, L0438:1, L0747:1, L0753:1 and L0758:1.
54	HMWJF53	758158	64	H0255:7, H0318:5, H0620:5, L0754:5, L0766:4, L0666:4, S0358:3, H0457:3, H0135:3, L0776:3, L0809:3, H0696:3, S012:3, H0624:2, H0295:2, H0254:2, H0662:2, H0402:2, H0305:2, S0132:2, L0717:2, L0021:2, H0617:2, H0673:2, L0769:2, L0638:2, L0796:2, L0667:2, L0662:2, L0653:2, L0783:2, L0663:2, S0126:2, H0539:2, H0521:2, S0044:2, S0027:2, L0745:2, L0747:2, L0755:2, L0587:2, H0352:2, H0170:1, H0556:1, H0657:1, H0341:1, S0212:1, S0418:1, S0360:1, S0410:1, H0339:1, H0549:1, S0222:1, H0441:1, H0331:1, H0486:1, H0427:1, H0575:1, T0048:1, H0581:1, H0052:1, H0545:1, H0150:1, H0570:1, H0569:1, L0163:1, H0083:1, H0355:1, H0252:1, H0039:1, T0023:1, H0124:1, H0090:1, H0413:1, H0560:1, H0561:1, S0372:1, H0509:1, H0652:1, S0144:1, S0422:1, L0762:1, L0770:1, L0761:1, L0373:1, L0372:1, L0645:1, L0764:1, L0771:1, L0648:1, L0768:1, L0649:1, L0804:1, L0651:1, L0806:1, L0655:1, L0659:1, L0517:1, L0528:1, L0665:1, H0698:1, S0374:1, L0438:1, H0684:1, H0658:1, H0670:1, S0328:1, S0380:1, H0134:1, S0406:1, L0743:1, L0749:1, L0750:1, L0779:1, L0759:1, S0031:1, H0445:1, H0653:1, S0194:1, S0276:1, H0542:1 and S0460:1.
55	HNECL22	799541	65	AR218:10, AR219:10, AR185:10, AR277:9, AR283:8, AR282:8, AR039:8, AR089:8, AR316:7, AR055:7, AR096:7, AR104:7, AR240:7, AR299:6, AR313:6, AR060:5, AR300:5 L0748:54, L0766:20, L0754:18, H0179:12, L0777:12, L0750:11, L0749:10, S0116:9, H0271:9, L0761:9, H0031:8, L0794:8, H0144:8, L0744:8, H0457:7, S0356:6, H0393:6, H0013:6, L0438:6, L0743:6, L0751:6, L0745:6, L0779:6, L0758:6, H0421:5, L0805:5, H0436:5, H0305:4, H0599:4, H0050:4, L0769:4, L0646:4, L0771:4, L0803:4, L0776:4, L0809:4, S0428:4, L0603:4, H0662:3, S0358:3, S0045:3, H0747:3, H0549:3, H0497:3, S0474:3, H0674:3, H0591:3, H0625:3, S0422:3, L0800:3, L0773:3, L0792:3, L0666:3, S0052:3, S0028:3, L0759:3, H0542:3, H0556:2, H0341:2, H0402:2, S0354:2, S0376:2, S0046:2, H0559:2, H0575:2, H0590:2, H0581:2, H0024:2, H0266:2, H0553:2, H0032:2, H0673:2, H0087:2, H0264:2, H0100:2, H0494:2, H0529:2, L0774:2, L0493:2, L0659:2, L0790:2, L0664:2, H0518:2, S0044:2, L0747:2, L0780:2, L0752:2, L0605:2, L0599:2, L0593:2, H0721:2, H0171:1, L3642:1, L3644:1, S0114:1, H0583:1, L0785:1, H0419:1, H0255:1, H0589:1, H0638:1, H0125:1, S0418:1, S0444:1, H0151:1, S0476:1, H0619:1, S0626:1, H0261:1, H0431:1, H0392:1, H0069:1, H0075:1, H0635:1, T0070:1, H0156:1, H0618:1, S0010:1, H0318:1, H0310:1, H0052:1, H0251:1, T0110:1, H0046:1, H0439:1, H0086:1, H0081:1, H0057:1, H0051:1, H0375:1, H0109:1, H0416:1, S0318:1, S0314:1, H0030:1, H0111:1, L0455:1, H0040:1, H0056:1, H0623:1, T0041:1, T0042:1, S0210:1, S0002:1, S0426:1, L0598:1, L0641:1, L0764:1, L0768:1, L0807:1, L0514:1, L0658:1, L0783:1, L5623:1, L0788:1, L0663:1, L0665:1, S0374:1, H0519:1, S0122:1, H0659:1, H0658:1, H0666:1, H0672:1, S0328:1, H0521:1, H0522:1, S0406:1, H0555:1, H0478:1, H0727:1, L0742:1, L0755:1, L0731:1, S0011:1, S0026:1, H0543:1, H0423:1, H0422:1 and H0506:1.
56	HNFAC50	815676	66	AR055:8, AR277:6, AR060:5, AR282:5, AR299:5, AR283:5, AR039:5, AR240:5, AR104:4, AR300:4, AR218:4, AR089:4, AR185:4, AR096:3, AR316:3, AR313:2, AR219:1 L0769:5, L0756:4, S0444:3, H0624:2, S0408:2, H0587:2, L0764:2, L0766:2, H0170:1, H0497:1, H0333:1, H0156:1, L0022:1, H0271:1, S0344:1, L0637:1, L0772:1, L0773:1, L0662:1, L0775:1, L0809:1, L0791:1, L0663:1, H0144:1, S0374:1, L3811:1, H0593:1, H0660:1, H0648:1, H0672:1, H0696:1, L0749:1, L0750:1, L0779:1, L0752:1, L0755:1, L0599:1, L0601:1 and H0667:1.
57	HNGEA34	815678	67	AR055:6, AR060:5, AR240:4, AR218:4, AR282:3, AR300:3, AR104:3, AR277:3, AR283:3, AR185:3, AR089:3, AR096:2,

58	HNGIV64	561572		AR316:2, AR219:2, AR039:2, AR313:2, AR299:2 H0393:1 and S0052:1. AR185:8, AR039:8, AR060:8, AR313:7, AR055:7, AR096:6, AR300:6, AR218:6, AR299:6, AR277:6, AR316:5, AR104:5, AR283:4, AR282:3, AR219:1 S0052:1
59	HNGKT41	836061	69	AR316:11, AR055:6, AR060:6, AR277:5, AR300:5, AR282:5, AR104:4, AR240:4, AR185:4, AR218:3, AR283:3, AR313:3, AR039:3, AR089:3, AR219:3, AR096:2, AR299:2 S0428:1
60	HNGNO53	836063	70	AR055:7, AR060:6, AR240:5, AR300:5, AR218:5, AR185:4, AR283:4, AR277:4, AR089:4, AR104:3, AR316:3, AR096:3, AR219:2, AR313:2, AR039:2, AR282:1 S0428:2 and L0439:1.
61	HNHCT47	634691	71	AR313:39, AR039:38, AR219:29, AR218:24, AR299:23, AR185:22, AR096:21, AR089:20, AR300:17, AR316:15, AR060:14, AR277:14, AR104:13, AR055:12, AR240:11, AR282:10, AR283:8 S0053:2 and S0046:1.
62	HNHKT74	777856	72	S0216:1
63	HORBS82	638293	73	H0706:2, L0809:2, S0360:1, L0623:1, H0122:1, H0041:1, H0095:1, H0292:1, H0424:1, S0364:1, L0794:1, L0787:1, L0663:1, H0780:1, H0435:1, L0743:1, L0747:1 and L0731:1.
64	HOUDE92	580866	74	H0052:17, L0745:11, L0748:10, H0547:7, L0439:7, L0755:6, L0771:5, L0774:5, L0662:4, L0746:4, L0777:4, S0474:3, L0163:3, H0059:3, H0100:3, L0775:3, L0741:3, H0261:2, H0333:2, H0194:2, H0545:2, H0012:2, H0617:2, H0135:2, L0770:2, L0665:2, L0438:2, H0520:2, L0747:2, L0752:2, L0753:2, S0040:1, L0717:1, H0437:1, H0550:1, S0616:1, H0497:1, H0574:1, H0599:1, H0575:1, H0618:1, H0253:1, H0041:1, H0620:1, H0373:1, H0188:1, H0124:1, H0068:1, H0040:1, H0561:1, S0448:1, S0210:1, L0763:1, L0644:1, L0767:1, L0768:1, L0375:1, L0651:1, L0659:1, L0540:1, L5622:1, H0144:1, H0593:1, S0126:1, H0539:1, S0152:1, H0694:1, S0390:1, S0028:1, L0749:1, L0786:1, L0780:1, L0731:1, L0757:1, L0758:1, S0436:1, L0592:1 and S0276:1.
65	HOUFS04	771564	75	AR218:41, AR219:38, AR096:23, AR185:23, AR277:22, AR299:22, AR282:20, AR055:18, AR316:17, AR039:16, AR089:14, AR240:13, AR283:13, AR104:12, AR300:10, AR313:10, AR060:10 L0745:15, S0414:6, H0351:5, H0013:5, S0422:5, L0803:5, H0144:4, H0413:3, H0519:3, L0754:3, L0759:3, S0242:3, H0624:2, H0580:2, S0045:2, L3655:2, H0421:2, H0375:2, H0428:2, H0553:2, L0598:2, L0775:2, L5622:2, L0666:2, L0664:2, L0665:2, H0520:2, H0547:2, S0126:2, H0672:2, S0380:2, H0521:2, L0743:2, L0744:2, L0605:2, H0171:1, H0556:1, H0685:1, S0040:1, S0114:1, H0657:1, S0212:1, S0444:1, H0733:1, H0734:1, H0749:1, S0132:1, H0619:1, L3388:1, H0411:1, S0278:1, H0549:1, S0222:1, L3816:1, H0486:1, S0280:1, H0575:1, L0105:1, H0581:1, H0052:1, H0545:1, H0594:1, S0628:1, H0687:1, S0250:1, H0031:1, S0364:1, L0455:1, H0124:1, H0591:1, H0038:1, S0450:1, L0763:1, L0638:1, L0637:1, L0662:1, L0794:1, L0649:1, L0654:1, L0382:1, L0792:1, L3811:1, L3824:1, L3828:1, H0435:1, H0518:1, H0696:1, H0436:1, S0432:1, S0390:1, S0037:1, S014:1, S0028:1, S0124:1, L0751:1, L0756:1, L0779:1, L0777:1, L0780:1, L0752:1, L0755:1, S0031:1, L0599:1, S0196:1, H0423:1, H0422:1 and H0721:1.
66	HOUHI25	888279	76	AR219:18, AR218:16, AR055:8, AR104:6, AR096:5, AR316:5, AR039:5, AR300:5, AR060:4, AR277:4, AR089:3, AR299:3, AR185:3, AR283:3, AR282:2, AR240:2, AR313:1 S0436:7, H0551:6, L2985:5, H0599:5, L0805:5, L0756:5, L0758:5, L0759:5, L0754:4, L0747:4, L3655:3, H0545:3, S0003:3, L0375:3, H0144:3, L0755:3, S0442:2, L3649:2, S0045:2, L3816:2, H0013:2, L0471:2, H0373:2, H0051:2, H0560:2, S0422:2, L0768:2, L0803:2, L0650:2, L0659:2, L0438:2, L0439:2, L0740:2, L0750:2, L0779:2, L0757:2, S0242:2, H0739:1, H0624:1, S0040:1, S0342:1, S0116:1, S0212:1, S0444:1, H0747:1, L3280:1, H0357:1, H0587:1, L0021:1, S0010:1, L0105:1, S0474:1, H0544:1, H0046:1, S0051:1, H0266:1, H0622:1, H0032:1, H0388:1, H0598:1, H0413:1, S0438:1, H0641:1, S0002:1, L0770:1, L3904:1, L0662:1, L0776:1, L0809:1, L0519:1, L5622:1, L5623:1,

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67	HPCAL26	762822	77	L0659:1, S0126:1, L0731:1, S0192:1, L0666:9, L0777:7, T0049:5, S0358:5, L0771:5, L0757:5, S0360:4, S0440:4, L0740:4, L0758:4, S0212:3, S0356:3, S0046:3, H0369:3, H0545:3, L0662:3, L0774:3, L0809:3, H0519:3, L0752:3, S0011:3, H0295:2, H0662:2, S0468:2, H0012:2, H0024:2, H0356:2, H0616:2, H0268:2, H0412:2, L0646:2, L0803:2, S0013:2, L0754:2, L0747:2, L0759:2, S0040:1, S0418:1, S0442:1, S0376:1, H0676:1, L0717:1, H0550:1, S0222:1, H0574:1, L0021:1, H0575:1, H0036:1, H0590:1, H0618:1, T0048:1, H0309:1, H0596:1, T0110:1, H0546:1, H0046:1, H0123:1, H0014:1, S0003:1, S0022:1, H0428:1, H0622:1, H0031:1, H0673:1, L0455:1, H0316:1, H0598:1, H0163:1, H0038:1, H0433:1, H0413:1, T0069:1, S0438:1, H0633:1, H0647:1, S0210:1, L0770:1, L0769:1, L0768:1, L0794:1, L0519:1, L0789:1, L0790:1, L0664:1, L0665:1, H0144:1, S0330:1, S0136:1, H0696:1, S014:1, S0206:1, L0751:1, L0749:1, L0756:1, L0779:1, S0031:1, S0242:1, S0194:1 and S0276:1.
68	HPEBA84	753957	78	AR313:5, AR240:4, AR299:4, AR185:4, AR300:4, AR060:4, AR055:3, AR089:3, AR096:2, AR282:2, AR039:2, AR218:2, AR316:2, AR277:1, AR219:1, AR104:1 L0591:2, L3643:1, S0420:1, L3388:1, H0057:1, H0166:1, L0648:1, L0518:1, L0809:1, L0519:1, L0754:1 and L0599:1.
69	HSAVA08	580870	79	AR313:39, AR039:39, AR299:18, AR089:17, AR096:17, AR185:16, AR277:16, AR300:16, AR104:12, AR316:12, AR240:10, AR219:10, AR218:9, AR060:9, AR282:9, AR055:8, AR283:5 S0114:2
70	HSAX04	812178	80	AR299:18, AR219:18, AR104:17, AR218:17, AR277:17, AR313:16, AR316:15, AR283:14, AR055:13, AR096:13, AR039:12, AR240:12, AR060:12, AR282:11, AR089:11, AR185:10, AR300:10 L0731:6, H0265:4, L0483:4, H0424:4, H0253:3, H0318:3, L0769:3, L0774:3, L0776:3, S0037:3, L0742:3, L0750:3, L0755:3, S0360:2, H0581:2, H0266:2, H0213:2, H0124:2, H0413:2, L0766:2, L0659:2, L0809:2, S014:2, L0749:2, L0757:2, L0758:2, L0759:2, L0596:2, L0595:2, H0543:2, H0422:2, H0686:1, H0685:1, S0040:1, H0295:1, H0294:1, S0430:1, H0638:1, S0418:1, S0420:1, S0354:1, S0358:1, S0376:1, S0045:1, H0586:1, H0497:1, H0333:1, H0486:1, H0069:1, H0575:1, H0618:1, H0052:1, H0085:1, H0009:1, S0051:1, H0083:1, H0284:1, H0428:1, H0417:1, H0553:1, H0628:1, H0038:1, H0280:1, H0494:1, H0625:1, S0150:1, S0426:1, L0667:1, L0646:1, L0764:1, L0773:1, L0648:1, L0767:1, L0768:1, L0375:1, L0806:1, L0519:1, L0666:1, L0663:1, H0698:1, H0689:1, H0539:1, H0518:1, S0027:1, S0028:1, L0747:1, L0752:1, H0707:1, L0597:1, L0581:1, L0361:1, H0653:1, H0542:1 and H0506:1.
71	HSKDR27	580874	81	AR055:9, AR104:9, AR218:7, AR060:7, AR299:6, AR185:6, AR039:6, AR240:5, AR089:5, AR219:5, AR300:5, AR283:5, AR316:4, AR313:4, AR096:3, AR277:3, AR282:2 S0027:95, S0192:54, S3014:53, S0126:42, S0040:35, H0424:23, S0028:22, S0037:19, S3012:16, H0213:13, T0006:12, H0250:11, S0032:11, L0744:11, T0040:10, H0124:10, H0429:10, L0740:10, L0588:10, L0754:9, H0545:8, H0280:8, S0194:8, S0196:7, H0392:6, T0039:6, H0150:6, H0039:6, S0206:6, L0743:6, L0731:6, S0342:5, S0212:5, S0045:5, H0486:5, H0575:5, H0014:5, H0090:5, H0551:5, H0100:5, S0044:5, S0011:5, H0255:4, H0318:4, H0271:4, S0022:4, H0031:4, H0181:4, H0032:4, H0038:4, T0067:4, S0124:4, L0747:4, L0749:4, H0402:3, H0309:3, H0046:3, S0250:3, H0068:3, H0087:3, H0059:3, S0142:3, S0053:3, H0419:2, S0116:2, S0408:2, S0132:2, S0278:2, S0222:2, H0331:2, T0060:2, H0069:2, H0427:2, H0599:2, T0082:2, H0253:2, H0546:2, H0086:2, H0123:2, H0024:2, H0015:2, H0510:2, H0428:2, T0023:2, H0163:2, H0063:2, H0509:2, L0772:2, L0805:2, S0052:2, H0547:2, H0518:2, L0748:2, L0751:2, L0745:2, L0750:2, L0777:2, L0755:2, L0757:2, H0445:2, L0590:2, L0599:2, S0026:2, S0242:2, H0171:1, H0265:1, H0716:1, H0294:1, S0298:1, H0662:1, H0450:1, S0360:1, H0329:1, S0046:1, H0411:1, S6022:1, H0431:1, H0357:1, H0455:1,

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72	HSQBF66	560726	82	AR313:16, AR039:13, AR089:11, AR299:10, AR185:9, AR277:8, AR055:8, AR060:8, AR240:8, AR096:8, AR300:8, AR104:7, AR316:7, AR282:7, AR218:6, AR283:5, AR219:3 S0026:1
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74	HSWBE76	751308	84	AR277:11, AR282:11, AR283:10, AR219:8, AR218:8, AR104:7, AR060:7, AR240:6, AR316:6, AR299:6, AR185:6, AR300:5, AR089:5, AR039:5, AR055:5, AR096:5, AR313:4 L0777:4, L0751:3, L0747:3, L0648:2, L0779:2, L0753:2, S0342:1, H0484:1, H0661:1, S0358:1, L0009:1, H0411:1, S0114:1, H0546:1, H0123:1, H0188:1, S0366:1, H0413:1, S0344:1, H0529:1, L0769:1, L0627:1, L0774:1, L0378:1, L0776:1, L0655:1, L0663:1, S0380:1, H0478:1, L0743:1, L0750:1 and S0196:1.
75	HT3BF49	838620	85	H0271:2, L0791:2, L0439:2, H0159:1, H0561:1, L0774:1, S0052:1 and L0779:1.
76	HTEEW69	764835	86	AR104:36, AR283:28, AR219:27, AR218:27, AR316:21, AR277:20, AR089:20, AR055:19, AR096:18, AR313:18, AR240:18, AR282:18, AR185:16, AR299:16, AR060:15, AR039:14, AR300:12 H0038:8, H0616:4, L0779:3, L0758:3, L0753:2, L0032:1, T0006:1, H0040:1, L0768:1 and H0547:1.
77	HTEHU59	840385	87	AR313:11, AR218:10, AR219:9, AR039:7, AR316:6, AR096:6, AR104:6, AR277:5, AR299:5, AR055:5, AR282:4, AR089:4, AR283:3, AR300:3, AR060:3, AR240:3, AR185:3 S0422:6, H0038:4, L0758:4, L0754:3, S0360:2, H0024:2, L0598:2, L0766:2, L0748:2, L0747:2, L0756:2, H0583:1, H0341:1, S0418:1, L0005:1, H0741:1, H0437:1, H0369:1, H0581:1, H0194:1, S0050:1, H0271:1, H0428:1, T0006:1, H0068:1, H0412:1, H0056:1, H0494:1, S0426:1, L0772:1, L0646:1, H0662:1, L0803:1, L0806:1, L0776:1, L0655:1, L0789:1, L0792:1, H0144:1, S0374:1, H0670:1, S0026:1 and S0192:1.
78	HTEMQ17	840387	88	AR282:6, AR055:6, AR060:5, AR218:4, AR283:4, AR300:3, AR299:3, AR316:3, AR039:3, AR185:3, AR104:2, AR089:2, AR219:2, AR313:2, AR096:2, AR240:1 L0748:6, L0766:4, H0038:3, H0616:3, H0056:2, H0529:2, H0519:2, H0624:1, H0662:1, S0418:1, S0360:1, H0749:1, H0013:1, H0581:1, S0388:1, H0266:1, H0591:1, H0087:1, H0413:1, H0561:1, S0438:1, S0422:1, L0520:1, L0769:1, L0794:1, L0775:1, L0666:1, L0663:1, H0547:1, S0152:1, L0740:1, L0777:1, L0753:1, L0758:1, L0608:1 and H0542:1.
79	HTGBK95	834490	89	AR277:83, AR313:74, AR219:72, AR283:69, AR316:57, AR039:49, AR218:49, AR089:48, AR299:46, AR282:42, AR104:42, AR096:42, AR185:40, AR240:39, AR055:37, AR300:32, AR060:32 L0777:5, S0444:3, L0766:3, L0803:3, L0439:3,

80	HTLEM16	779133	90	S0360:2, L0598:2, L0666:2, L0748:2, T0049:1, S0134:1, S0116:1, S0408:1, L0717:1, H0586:1, H0486:1, H0575:1, H0510:1, H0553:1, H0560:1, S0422:1, L0763:1, L0769:1, L0521:1, L0767:1, L0768:1, L0775:1, L0663:1, S0374:1, L0438:1, H0520:1, H0682:1, S0328:1, S0406:1, L0740:1, S0192:1 and H0543:1. AR104:96, AR219:74, AR277:67, AR283:59, AR218:52, AR185:51, AR089:49, AR316:46, AR096:44, AR240:44, AR313:42, AR055:40, AR299:37, AR282:37, AR060:33, AR039:33, AR300:24 L0439:31, L0741:24, H0056:13, L0748:12, H0052:9, H0521:9, L0776:8, L0744:8, L0438:7, L0754:7, S0474:6, L0766:6, L0742:6, L0731:6, L0750:5, S0278:4, L5566:4, L0665:4, H0522:4, H0556:3, H0716:3, H0657:3, S0358:3, H0580:3, H0599:3, S0049:3, H0009:3, H0553:3, H0641:3, S0142:3, L0764:3, L0659:3, L0666:3, S0126:3, L0751:3, H0717:2, H0656:2, S0029:2, S0420:2, S0360:2, S0007:2, H0497:2, H0486:2, H0618:2, H0253:2, H0581:2, H0046:2, S0388:2, T0010:2, H0039:2, H0424:2, L0456:2, S0036:2, H0135:2, H0551:2, H0623:2, H0494:2, S0002:2, L0770:2, L0796:2, L5575:2, L5565:2, L0761:2, L0662:2, L0650:2, L0383:2, H0682:2, L0758:2, S0434:2, L0596:2, L0581:2, S0242:2, S0114:1, H0583:1, L0422:1, S0116:1, H0662:1, H0305:1, S0418:1, L0005:1, S0444:1, S0046:1, S0476:1, H0645:1, H0437:1, H0261:1, H0392:1, H0600:1, H0586:1, H0574:1, L0623:1, H0013:1, H0250:1, H0427:1, H0002:1, H0575:1, T0082:1, H0590:1, S0010:1, H0390:1, T0048:1, H0318:1, H0421:1, H0251:1, H0232:1, H0546:1, H0150:1, H0041:1, H0178:1, H0569:1, H0620:1, H0051:1, S0051:1, H0510:1, H0416:1, H0188:1, S0312:1, S0314:1, H0622:1, H0213:1, H0031:1, L0143:1, H0032:1, L0455:1, S0366:1, H0038:1, H0087:1, H0264:1, H0268:1, H0022:1, H0560:1, H0625:1, H0561:1, S0438:1, H0509:1, H0633:1, H0649:1, S0144:1, S0208:1, H0529:1, L0769:1, L0637:1, L0667:1, L5568:1, L0774:1, L0375:1, L0805:1, L0653:1, L0654:1, L0661:1, L0807:1, L0527:1, L0382:1, L0809:1, L0793:1, S0006:1, S0428:1, S0053:1, S0310:1, L0352:1, H0547:1, H0684:1, H0670:1, H0660:1, S0152:1, H0696:1, S0406:1, H0555:1, H0436:1, S3014:1, L0743:1, L0745:1, L0747:1, L0749:1, L0756:1, L0753:1, L0755:1, H0445:1, S0436:1, L0485:1, H0667:1, H0216:1, H0543:1, H0422:1 and H0008:1. L0779:5, L0731:4, L0593:4, H0046:3, L0776:3, L0666:3, H0031:2, L0772:2, L0774:2, L0805:2, H0670:2, L0439:2, L0754:2, L0777:2, L0758:2, L0590:2, T0002:1, L0717:1, H0632:1, L0622:1, T0082:1, H0581:1, H0263:1, T0115:1, H0597:1, L0471:1, H0012:1, H0620:1, H0163:1, T0067:1, L0770:1, L0637:1, L0388:1, L0657:1, L0382:1, L0664:1, S0126:1, H0660:1, S0378:1, H0521:1, L0747:1, L0750:1, L0756:1, L0752:1, L0755:1, S0031:1, L0599:1 and L0603:1. AR104:20, AR185:11, AR055:11, AR060:10, AR089:10, AR299:10, AR240:9, AR277:8, AR282:8, AR313:7, AR300:7, AR316:7, AR283:5, AR096:5, AR218:4, AR219:4, AR039:3 H0264:1 AR039:5, AR277:4, AR300:4, AR282:3, AR316:3, AR096:3, AR218:3, AR299:3, AR060:2, AR055:2, AR283:2, AR185:2, AR104:2, AR313:2, AR089:1, AR240:1 H0677:19, L0759:6, L0748:5, H0040:4, L0438:3, L0754:3, L0750:3, L0777:3, H0255:2, H0617:2, H0038:2, H0529:2, L0769:2, L0761:2, L0662:2, L0666:2, S0406:2, L0749:2, L0758:2, L0595:2, H0265:1, H0556:1, H0717:1, S0134:1, H0650:1, H0657:1, S0358:1, S0444:1, S0410:1, S0045:1, H0411:1, H0392:1, L0468:1, H0587:1, H0013:1, H0069:1, H0635:1, H0575:1, H0618:1, H0581:1, H0564:1, H0569:1, S0028:1, H0266:1, H0252:1, H0615:1, H0039:1, H0031:1, H0634:1, H0100:1, H0494:1, H0334:1, H0561:1, S0150:1, S0422:1, L0667:1, L0646:1, L0800:1, L0771:1, L0661:1, L0809:1, L0790:1, L0792:1, L0663:1, L0665:1, S0374:1, H0547:1, H0519:1, H0593:1, H0672:1, H0518:1, H0521:1, H0555:1, H0436:1, L0439:1, L0779:1, L0731:1 and L0757:1. AR218:21, AR219:20, AR089:15, AR300:14, AR316:14, AR185:13, AR313:13, AR277:13, AR282:13, AR039:12, AR299:11, AR096:11, AR055:10, AR104:8, AR240:7, AR060:7, AR283:6
81	HTNBK13	831967	91	
82	HTODN35	570901	92	
83	HTPDU17	840596	93	
84	HTTDN24	766485	94	

85	HTTEE41	840950	95	AR219:84, AR218:59, AR316:43, AR313:32, AR104:24, AR089:24, AR185:24, AR039:23, AR096:23, AR299:21, AR055:20, AR060:17, AR282:14, AR300:14, AR283:11, AR240:11, AR277:10, H0040:17, H0251:14, L0758:10, L0748:8, L0731:8, H0494:7, L0666:7, H0144:7, H0659:7, L0747:7, L0749:7, H0038:6, H0529:6, L0770:6, L0662:6, L0659:6, H0013:5, H0318:5, H0616:5, S0440:5, L0775:5, L0776:5, H0519:5, L0588:5, L0592:5, H0341:4, S0360:4, H0412:4, L0663:4, H0547:4, L0754:4, L0595:4, H0542:4, H0543:4, H0423:4, H0171:3, H0657:3, H0656:3, S0045:3, L3388:3, H0581:3, S0049:3, T0110:3, H0046:3, H0090:3, H0591:3, H0551:3, H0100:3, H0022:3, H0625:3, H0633:3, S0422:3, L0375:3, L0664:3, H0682:3, S0406:3, L0740:3, H0556:2, H0241:2, H0638:2, S0418:2, L0005:2, S0442:2, S0376:2, H0722:2, H0393:2, L0717:2, S0222:2, H0574:2, H0486:2, T0040:2, L0471:2, S0051:2, S0003:2, H0252:2, L0483:2, T0006:2, H0031:2, H0032:2, H0124:2, H0634:2, H0264:2, T0042:2, S0150:2, H0646:2, L0763:2, L0637:2, L0646:2, L0374:2, L0764:2, L0768:2, L0653:2, L0665:2, H0593:2, H0435:2, H0658:2, H0539:2, S0152:2, L3832:2, H0521:2, S3014:2, S0027:2, S0028:2, L0439:2, L0750:2, L0777:2, S0436:2, L0596:2, L0608:2, L0604:2, L0594:2, L0362:2, S0026:2, H0667:2, S0452:2, H0506:2, L0411:1, H0624:1, H0170:1, H0395:1, H0265:1, T0002:1, H0220:1, H0140:1, H0159:1, H0686:1, H0583:1, S0212:1, H0484:1, H0664:1, L0481:1, S0356:1, S0354:1, S0358:1, S0444:1, S0408:1, L3649:1, H0580:1, H0747:1, H0437:1, H0431:1, T0104:1, H0600:1, H0592:1, H0586:1, L3817:1, H0642:1, H0632:1, L2482:1, T0114:1, H0244:1, H0250:1, H0069:1, H0156:1, L0021:1, H0599:1, H0036:1, S0346:1, H0596:1, H0544:1, H0009:1, N0006:1, L0157:1, H0569:1, H0123:1, H0242:1, H0024:1, H0083:1, H0375:1, H0328:1, H0615:1, H0428:1, H0039:1, H0622:1, H0213:1, H0553:1, L0142:1, H0628:1, H0674:1, H0388:1, L0456:1, H0708:1, H0068:1, H0598:1, S0036:1, H0135:1, H0087:1, H0380:1, H0413:1, H0056:1, L0351:1, T0041:1, H0334:1, H0561:1, H0366:1, S0448:1, S0294:1, H0130:1, H0641:1, H0649:1, S0208:1, S0002:1, S0426:1, L0520:1, L0631:1, L0769:1, L0638:1, L5565:1, L0667:1, L0772:1, L0372:1, L0641:1, L0626:1, L0794:1, L0766:1, L0381:1, L0650:1, L0651:1, L0806:1, L0655:1, L0807:1, L0657:1, L0636:1, L0518:1, L0782:1, L0382:1, L0809:1, L3391:1, L2263:1, L2259:1, L2262:1, L0565:1, H0693:1, L3827:1, H0520:1, S0126:1, H0689:1, H0670:1, H0660:1, H0666:1, H0648:1, L0602:1, H0710:1, H0518:1, S0176:1, H0134:1, H0555:1, H0436:1, H0478:1, H0631:1, L0779:1, L0752:1, S0434:1, L0605:1, L0591:1, L0599:1, H0665:1, S0196:1, L2368:1, H0008:1 and H0352:1.
86	HTXJD85	840391	96	AR313:38, AR039:32, AR096:17, AR185:17, AR300:17, AR277:16, AR299:15, AR089:15, AR240:12, AR316:10, AR219:10, AR104:9, AR060:8, AR055:8, AR218:8, AR282:6, AR283:4, H0556:2, L0638:1, L0748:1 and L0439:1.
87	HUVDJ48	564853	97	AR055:6, AR060:5, AR283:5, AR039:5, AR185:4, AR096:4, AR240:4, AR104:4, AR299:4, AR300:3, AR089:3, AR316:3, AR313:3, AR282:3, AR218:2, AR277:2, AR219:2, H0393:1, H0056:1 and L0662:1.
88	HWBBU75	780360	98	L0665:4, H0457:3, H0264:3, L0766:3, H0521:3, L0745:3, H0556:2, H0580:2, S0352:2, L0761:2, L0806:2, L0789:2, L0748:2, H0542:2, H0255:1, S0278:1, H0581:1, H0271:1, H0719:1, H0413:1, H0494:1, S0002:1, S0426:1, L0769:1, L0774:1, H0660:1, L0750:1, L0752:1, L0753:1 and S0424:1.
89	HWHPB78	740778	99	H0437:2, L0769:2, S0028:2, L0439:2, S0436:2, H0556:1, H0125:1, S0420:1, H0619:1, H0587:1, H0635:1, H0253:1, H0318:1, H0744:1, H0052:1, H0009:1, H0172:1, H0266:1, H0135:1, H0494:1, L3905:1, L0438:1, L3828:1, H0547:1, H0539:1, H0521:1, S0037:1, L0593:1, H0506:1 and H0008:1.
90	HWLBO67	834315	100	S0374:1
91	HWLGP26	834770	101	AR313:10, AR039:7, AR096:6, AR316:5, AR299:4, AR240:4, AR300:3, AR277:3, AR089:3, AR060:2, AR185:2, AR282:2, AR055:2, AR218:1, AR283:1, L0766:5, L0803:5, L0794:3, S0410:2, H0551:2, H0435:2, L0756:2, L0731:2, H0585:1,

92	HILCA24	869856	102	S0212:1, L0534:1, S0442:1, S0354:1, H0735:1, H0486:1, H0014:1, H0354:1, H0644:1, H0135:1, H0647:1, L0369:1, L0640:1, L0763:1, L0770:1, L3905:1, L0646:1, L0771:1, L0804:1, L0784:1, L0528:1, L0789:1, L0790:1, L0792:1, L3827:1, H0658:1, L0749:1, L0758:1 and S0436:1.
93	HE2CA60	888705	103	AR316:4, AR282:2, AR096:1, AR299:1, AR039:1 L0748:4, H0090:2, L0659:2, H0521:2, L0777:2, L0608:2, H0543:2, T0002:1, S0114:1, L3658:1, S0358:1, S0408:1, L3649:1, T0109:1, H0581:1, H0622:1, H0031:1, H0644:1, S0002:1, L0657:1, L0526:1, L0789:1, L0664:1, S0380:1, H0522:1, L0749:1 and L0779:1. AR313:86, AR299:44, AR277:42, AR283:37, AR039:37, AR316:36, AR218:34, AR096:34, AR219:34, AR089:32, AR185:32, AR104:30, AR282:23, AR300:23, AR055:22, AR060:16, AR240:16 H0305:16, L0777:11, L0471:10, S0422:9, L0766:9, H0624:8, H0013:7, H0170:6, L2551:6, H0046:6, L0665:6, L0598:5, L0662:5, L0776:5, H0547:5, L0758:5, L0589:5, H0171:4, L0659:4, L0666:4, L0663:4, L0756:4, L0731:4, S0358:3, L2744:3, L3655:3, H0581:3, H0457:3, S0406:3, L0744:3, L0439:3, L0752:3, S0436:3, H0542:3, H0543:3, L3643:2, H0650:2, H0657:2, S0116:2, S0442:2, S0354:2, L0717:2, S0414:2, H0486:2, T0040:2, H0318:2, H0421:2, H0428:2, H0553:2, H0090:2, H0040:2, H0063:2, H0641:2, L0769:2, L0761:2, L0764:2, L0650:2, L0774:2, L0805:2, L0657:2, H0144:2, L3811:2, L3832:2, H0521:2, S0404:2, L0741:2, L0740:2, L0747:2, L0759:2, S0434:2, L0362:2, H0685:1, S0218:1, L0785:1, H0341:1, H0255:1, H0663:1, H0662:1, H0402:1, S0376:1, S0360:1, S0410:1, L3645:1, L3646:1, H0637:1, H0741:1, H0722:1, H0735:1, S0046:1, H0749:1, S0300:1, L2758:1, L2767:1, L3388:1, S0222:1, H0592:1, H0586:1, H0587:1, H0559:1, L3653:1, H0427:1, L0021:1, H0037:1, H0746:1, H0263:1, H0544:1, H0050:1, H0057:1, L0163:1, H0051:1, S0022:1, H0328:1, T0023:1, H0673:1, H0674:1, H0591:1, H0038:1, H0551:1, T0067:1, H0100:1, L0065:1, S0440:1, H0649:1, H0529:1, L0369:1, L0763:1, L0667:1, L0630:1, L0372:1, L0521:1, L0533:1, L0775:1, L0651:1, L0806:1, L0655:1, L0661:1, L0807:1, L0656:1, L0809:1, L3872:1, L0790:1, L0664:1, L2655:1, L3663:1, S0374:1, L2706:1, H0520:1, H0435:1, H0660:1, H0672:1, S0328:1, H0539:1, S0380:1, H0753:1, S0004:1, H0696:1, L0748:1, L0754:1, L0750:1, L0753:1, S0031:1, H0444:1, L0588:1, L0605:1, L0485:1, H0216:1, S0242:1, H0423:1, S0458:1 and H0721:1.
94	HPWTF23	844775	104	AR240:84, AR089:51, AR299:38, AR096:37, AR039:36, AR313:34, AR185:24, AR300:22, AR316:22, AR282:19, AR283:15, AR104:14, AR055:10, AR060:10, AR277:6, AR218:2 S0474:47, H0710:28, L0747:18, L0659:17, H0656:13, H0436:10, H0271:9, L0751:9, H0581:8, H0179:8, H0063:8, L0731:8, L0599:8, H0740:7, L0756:7, H0650:6, L0662:6, H0555:6, S0354:5, H0728:5, H0733:5, H0734:5, H0036:5, H0590:5, H0052:5, L0770:5, S0428:5, S0374:5, L0439:5, L3643:4, H0717:4, H0747:4, H0393:4, S0222:4, H0156:4, H0309:4, S0312:4, S0314:4, H0090:4, H0591:4, L0637:4, L0761:4, L0776:4, L0783:4, L0438:4, L0757:4, H0543:4, H0716:3, H0662:3, H0402:3, H0619:3, H0392:3, H0575:3, H0004:3, H0673:3, S0364:3, H0135:3, H0059:3, H0494:3, L0667:3, L0764:3, L0803:3, L0775:3, L0666:3, L3811:3, H0670:3, L0744:3, L0750:3, L0779:3, L0758:3, L0759:3, S0436:3, H0556:2, L3644:2, H0713:2, S0624:2, L0666:3, L3811:3, H0670:3, L0744:3, L0750:3, L0779:3, L0758:3, L0759:3, S0436:3, H0556:2, L3644:2, H0713:2, S0624:2, H0341:2, S0282:2, S0442:2, S0376:2, S0360:2, H0580:2, H0329:2, H0749:2, H0645:2, H0369:2, H0486:2, S0280:2, H0042:2, H0421:2, H0545:2, H0457:2, H0620:2, H0014:2, S0051:2, T0010:2, S0340:2, H0031:2, H0644:2, H0383:2, H0674:2, H0551:2, H0264:2, H0488:2, T0004:2, S0438:2, H0130:2, H0647:2, S0422:2, L0766:2, L0655:2, L0517:2, L0518:2, L0809:2, L0647:2, H0547:2, H0660:2, S0044:2, S0027:2, S0028:2, L0748:2, L0754:2, L0745:2, H0445:2, S0434:2, L0596:2, L0588:2, H0506:2, H0170:1, S0134:1, L0414:1, L0785:1, S0212:1, H0255:1, S0358:1, S0444:1, L3649:1, H0637:1, H0729:1, H0730:1, H0741:1, H0208:1, S0222:1, H0550:1, H0609:1, H0586:1, H0333:1, T0060:1, H0427:1, L0021:1, L0022:1, S0010:1, S0346:1,

					L0105:1, H0318:1, H0597:1, H0150:1, L0471:1, H0011:1, S0362:1, H0373:1, S0388:1, H0354:1, H0099:1, H0594:1, H0266:1, H0416:1, H0188:1, S0318:1, S0334:1, H0687:1, S0338:1, H0252:1, H0213:1, H0553:1, H0111:1, H0617:1, H0169:1, H0163:1, T0067:1, L0435:1, L0564:1, S0440:1, H0509:1, S0150:1, H0646:1, H0652:1, L3815:1, L0371:1, L0769:1, L0771:1, L0649:1, L0774:1, L0375:1, L0651:1, L0378:1, L0805:1, L0606:1, L0657:1, L0384:1, L0529:1, L5623:1, L0793:1, L0664:1, S0216:1, H0144:1, H0723:1, H0593:1, H0689:1, H0659:1, H0672:1, S0328:1, H0539:1, H0518:1, H0521:1, H0696:1, H0134:1, L0612:1, H0732:1, S012:1, S0390:1, S0037:1, S0314:1, S0032:1, L0743:1, L0749:1, L0752:1, L0755:1, H0707:1, L0591:1, L0592:1, H0653:1, H0136:1, S0412:1 and H0721:1.
95	HGCAC19	851527	105		AR219:2, AR316:2, AR096:1 L0794:15, L0803:12, L0766:7, H0013:6, H0090:6, L0663:6, L0777:6, L0731:6, L0759:6, H0457:5, H0328:5, L0493:5, L0666:5, L0754:5, L0749:5, H0543:5, H0656:4, S0358:4, H0615:4, L0665:4, H0521:4, L0779:4, L0588:4, H0305:3, S0360:3, H0036:3, H0052:3, T0042:3, L0761:3, L0805:3, L0809:3, H0144:3, H0670:3, H0696:3, L0591:3, S0134:2, H0657:2, L3659:2, S0418:2, S0442:2, S0007:2, S0045:2, L0717:2, H0600:2, H0486:2, H0156:2, H0575:2, H0590:2, H0024:2, S0022:2, L0483:2, H0135:2, H0038:2, H0560:2, S0422:2, L0457:2, H0529:2, L0625:2, L0648:2, L0776:2, L0655:2, L0527:2, S0374:2, H0520:2, H0519:2, H0659:2, H0436:2, L0748:2, L0745:2, L0581:2, L0361:2, H0542:2, H0423:2, S0424:2, H0624:1, H0171:1, H0556:1, T0002:1, H0686:1, S0342:1, H0717:1, T0049:1, S0430:1, H0650:1, H0341:1, H0663:1, H0589:1, S0356:1, S0376:1, S0408:1, S0410:1, L2336:1, H0329:1, S0046:1, H0645:1, H0369:1, S0014:1, H0370:1, H0455:1, H0438:1, H0602:1, H0586:1, H0587:1, H0574:1, H0559:1, S0280:1, L0021:1, H0318:1, S0474:1, H0263:1, T0115:1, H0545:1, L0157:1, H0123:1, L0471:1, H0015:1, S0388:1, S0051:1, H0375:1, H0271:1, H0188:1, S0312:1, S0003:1, H0688:1, H0039:1, H0622:1, H0031:1, H0644:1, L0055:1, H0169:1, L0456:1, H0163:1, H0634:1, H0551:1, H0379:1, H0488:1, H0279:1, L0475:1, S0352:1, H0652:1, S0208:1, L0640:1, L0763:1, L0500:1, L0769:1, L0646:1, L0662:1, L0649:1, L0498:1, L0804:1, L0650:1, L0784:1, L0806:1, L0653:1, L0606:1, L0515:1, L0659:1, L0526:1, L0519:1, L0788:1, L0790:1, L0791:1, L0664:1, S0053:1, S0296:1, H0547:1, S0126:1, H0682:1, H0684:1, H0658:1, H0660:1, H0672:1, S0380:1, H0518:1, H0525:1, S0044:1, S0404:1, S0406:1, H0479:1, S0432:1, S3014:1, L0744:1, L0750:1, L0780:1, L0753:1, L0604:1, S0106:1, S0242:1, S0196:1, S0452:1 and H0506:1.
96	HEQBJ01	876546	106		AR277:12, AR283:11, AR219:11, AR316:10, AR089:10, AR218:9, AR104:9, AR055:8, AR282:8, AR313:8, AR096:7, AR300:7, AR185:7, AR299:7, AR240:7, AR060:5, AR039:5 S0360:3, H0619:3, H0673:2, L0438:2, H0685:1, S0444:1, H0544:1, H0266:1, H0163:1, L0770:1, L0646:1, L0768:1, L0766:1, L0803:1, L0776:1, S0152:1, S0027:1, L0439:1, L0747:1, L0777:1, L0752:1 and L0758:1.
97	HBJHT01	587262	107		AR313:10, AR039:8, AR299:6, AR300:5, AR185:5, AR277:5, AR060:4, AR096:4, AR089:4, AR316:4, AR240:3, AR104:3, AR282:2, AR218:2, AR283:2 L0667:2, S0114:1, H0351:1, H0318:1, H0615:1 and L0764:1.

Table 1C summarizes additional polynucleotides encompassed by the invention (including cDNA clones related to the sequences (Clone ID:), contig sequences (contig identifier (Contig ID:) contig nucleotide sequence identifiers (SEQ ID NO:X)), and genomic sequences (SEQ ID NO:B). The first column provides a unique clone identifier, "Clone ID:", for a cDNA clone related to each contig sequence. The second column provides the sequence identifier, "SEQ ID NO:X", for each contig sequence. The third column provides a unique contig identifier, "Contig ID:" for each contig sequence. The fourth column, provides a BAC identifier "BAC ID NO:A" for the BAC clone referenced in the corresponding row of the table. The fifth column provides the nucleotide sequence identifier, "SEQ ID NO:B" for a fragment of the BAC clone identified in column four of the corresponding row of the table. The sixth column, "Exon From-To", provides the location (i.e., nucleotide position numbers) within the polynucleotide sequence of SEQ ID NO:B which delineate certain polynucleotides of the invention that are also exemplary members of polynucleotide sequences that encode polypeptides of the invention (e.g., polypeptides containing amino acid sequences encoded by the polynucleotide sequences delineated in column six, and fragments and variants thereof).

Table 1C

cDNA Clone ID	SEQ ID NO:X	CONTIG ID:	BAC ID: A	SEQ ID NO:B	EXON From-To
HADMA77	14	783049	AC007944	221	1-3350
HADMA77	14	783049	AC018656	222	1-3349
HADMA77	14	783049	AC021874	223	1-3351 4529-4959 6110-6438
HADMA77	14	783049	AC007944	224	1-941
HADMA77	14	783049	AC018656	225	1-432
HADMA77	14	783049	AC018656	226	1-941
HADMB15	15	847116	AC026666	227	1-385 406-780
HADMB15	15	847116	AC026281	228	1-114 430-875 896-1262
HAGHR18	19	655435	AC009671	229	1-1134
HAQAI92	20	688037	AL118502	230	1-471 571-1561
HAQAI92	20	688037	AL161939	231	1-471 571-1561
HAQAI92	20	688037	AC004064	232	1-471 571-1561
HAQAI92	20	688037	AL118502	233	1-161
HAQAI92	20	688037	AL118502	234	1-285
HAQAI92	20	688037	AL161939	235	1-415

HAQAI92	20	688037	AL161939	236	1-285
HAQAI92	20	688037	AC004064	237	1-285
HAQAI92	20	688037	AC004064	238	1-415
HBAGD86	23	838799	AC016755	239	1-41 1648-1993 2035-3552 3554-6713
HBAGD86	23	838799	AC016755	240	1-161 696-809 2256-2753 6910-6991 7733-7857 9267-9458 10650-10734 11114-11562 11678-11801 12524-12817 14494-15914
HBAGD86	23	838799	AC016755	241	1-217
HBJAB02	25	837309	AC015651	242	1-35 159-252 410-783 786-830 953-1035 1452-1553 1651-2071 2161-2264 2352-2454 2494-2758 2847-3006 3135-3272 3477-4138 4907-5738 5972-6059 6132-6367 6650-6834 6915-7010 7091-7658 7662-9457 10122-10222 11415-11534 12386-12418 13253-13584 13635-13867 14881-15326 15851-16013 16529-16816 17430-17529 18140-18269 18634-18734 19189-19369 20434-21105

					21912-22008
HBJAB02	25	837309	AC015651	243	1-2097 5308-5495 5696-5742 5890-6249 7370-7525 7850-8236 8359-8463 8597-8770 8919-9028 9213-9353 9517-9639 9765-9874 9944-11023 11124-11219 11315-11613 11708-12241 12431-12666 12744-12802 12976-13087 13374-13914 14728-15500
HCEEE79	30	560609	AC006923	244	1-1044
HCEEE79	30	560609	AC006923	245	1-207
HCUCF89	32	637986	AC022554	246	1-1066
HCUCF89	32	637986	AC022554	247	1-692
HCUCF89	32	637986	AC022554	248	1-643
HCWAE64	33	535893	AL157935	249	1-1319 2024-2316 2937-2984 3126-3281 5595-5703 5788-6574 6667-6733 6788-6880 6962-7303 8111-11869 12019-12418 12420-12679 13140-13191
HCWAE64	33	535893	AL157935	250	1-1316
HCWAE64	33	535893	AL157935	251	1-309
HCWUL09	34	834722	AL138741	252	1-755
HCWUL09	34	834722	AL138741	253	1-555
HDPDI72	35	897277	AL139238	254	1-76 3170-3542 4724-5613 6598-6719 6954-7373 8256-8349 10408-11003
HDPDI72	35	897277	AL139238	255	1-279

HDPFY18	36	779450	AC011875	256	1-1880
HDPFY18	36	779450	AP000848	257	1-1470
HDPFY18	36	779450	AP000663	258	1-3332
HDTLM18	40	836057	AL049843	259	1-148 811-1104 2196-2793 2930-2991 3921-4047 6575-6627 8124-8659 8741-8843 9448-9886 10480-10524 10944-11103 13917-14450 14801-15344 16392-17295 18110-18311 20445-21421 21596-22268 23857-23968 24205-24585 24623-24701 25168-25575 28078-28391 28548-28707 29039-29839 30732-31495 32024-32487 32521-33216 34511-34647 35166-35720 36527-36797 36993-37125 38178-38288 39341-39646 41511-41570 42307-42873 42914-43014 43248-43465 43589-43690 43724-43909 44170-44333 44517-45130 45497-45961 46215-46842 47926-48126 49391-51961
HDTLM18	40	836057	AL049843	260	1-2071
HDTLM18	40	836057	AL049843	261	1-76 2590-2720 4185-4370

					7052-7178 7385-7428
HE8BQ49	42	589443	AC009225	262	1-1857
HEBFR46	45	847064	AC006483	263	1-70 282-644 789-4243
HEBFR46	45	847064	AC073481	264	1-2167 2174-3461
HEBFR46	45	847064	AC006483	265	1-344
HEBFR46	45	847064	AC006483	266	1-195
HFCDW95	47	847383	AC006388	267	1-3484
HFCDW95	47	847383	AC006388	268	1-1218
HFCDW95	47	847383	AC006388	269	1-158
HFIJA29	49	839206	AL031259	270	1-1291 1460-2067 2069-2908 3053-3754 4093-4182 4546-4650 5612-6170 6932-9872
HFIJA29	49	839206	AC009954	271	1-1294 1463-2070 2072-2911 3056-3757 4096-4185 4549-4653 5614-6172 6935-9878
HFIJA29	49	839206	AL031259	272	1-426
HFIJA29	49	839206	AL031259	273	1-829 1325-1871 2204-2663 3085-3251 4136-4238 4289-4388 4564-4997 5413-5945 8022-8401 8405-8923 10401-10602 10628-11074 15099-15200 15346-15984 18400-18491
HFIJA29	49	839206	AC009954	274	1-829 1325-1871 2204-2663 3087-3253 4262-4399 4571-5004 5420-5952

					8016-8395 8399-8917 10395-10596 10622-11056 15325-15963 18377-18495
HFIIA29	49	839206	AC009954	275	1-426
HFKFX64	50	566835	AP001203	276	1-870
HFKFX64	50	566835	AC025291	277	1-868
HFKFX64	50	566835	AC010798	278	1-868
HFKFX64	50	566835	AP001203	279	1-750
HFKFX64	50	566835	AC025291	280	1-750
HFKFX64	50	566835	AC010798	281	1-750
HGBER72	51	826710	AL157935	282	1-1319 2024-2316 2937-2984 3126-3281 5595-5703 5788-6574 6667-6733 6788-6880 6962-7303 8111-11869 12019-12418 12420-12679 13140-13191
HGBER72	51	826710	AL157935	283	1-1316
HGBER72	51	826710	AL157935	284	1-309
HGLBG15	53	701990	AC005082	285	1-252 2150-2418 2461-2695 2700-3101 3368-3776 6066-6250 7105-7363 8329-9740
HGLBG15	53	701990	AC073992	286	1-259 1225-2636
HGLBG15	53	701990	AC005082	287	1-546
HGLBG15	53	701990	AC073992	288	1-282
HHFEC39	54	609873	AL022726	289	1-819 1137-4813
HHFEC39	54	609873	AL022726	290	1-509
HHSDI53	55	862028	AP001456	291	1-1611 1654-2020 2187-2263
HHSDI53	55	862028	AL109936	292	1-1611 1654-2020 2186-2322 2673-3243 3291-3857 4276-4892

					5002-5380 8185-8499 8705-8842 10146-10298 12526-12652 12780-14327
HHSDI53	55	862028	AP001456	293	1-482
HHSDI53	55	862028	AL109936	294	1-188
HISBA38	56	561711	AL137020	295	1-1169
HISBA38	56	561711	AL359254	296	1-1169
HISBA38	56	561711	AL137020	297	1-702
HISBA38	56	561711	AL359254	298	1-702
HLYGE16	59	651339	AC025594	299	1-272 301-388 531-1439 1461-3200
HLYGE16	59	651339	AC073849	300	1-272 301-388 531-1439 1461-3200
HLYGE16	59	651339	AC025594	301	1-337
HLYGE16	59	651339	AC073849	302	1-337
HMCFH60	61	654853	AL122034	303	1-785 1072-3055
HMCFH60	61	654853	AC073394	304	1-326 1898-2079 2460-2702 4498-4586 5598-7296 7560-7669 8015-8460 8479-8539 8918-9242 10451-10975 13375-13521 13561-15769 16055-18038
HMCFH60	61	654853	AL160264	305	1-86 1101-2799 3063-3172 3518-3963 3982-4042 4421-4745 5954-6478 8877-9023 9063-11271 11557-13540
HMCFH60	61	654853	AC073394	306	1-309
HMCFH60	61	654853	AC073394	307	1-577
HMLAK10	62	562774	AP000817	308	1-1044
HMLAK10	62	562774	AC024177	309	1-1047
HMLAK10	62	562774	AC011009	310	1-1047

HMIBD93	63	634227	AC010913	311	1-3640
HMIBD93	63	634227	AC010913	312	1-495 593-668 670-1055 1578-1799 2445-2717 3103-3203 3284-3751 3841-4032 5093-5261 5443-5872 5922-6838 7633-8170 8304-8491 8968-9029 9888-10020 10479-10733 10807-10958 11020-11132 12080-12373 12464-12585 13223-13381 17379-17471 18572-19447
HMWJF53	64	758158	AC021016	313	1-739 792-852 1482-1572 1670-4387
HMWJF53	64	758158	AC021016	314	1-276
HNECL22	65	799541	AF216674	315	1-2837
HNECL22	65	799541	AC051642	316	1-2201
HNECL22	65	799541	AF216674	317	1-462
HNECL22	65	799541	AF216674	318	1-836
HNECL22	65	799541	AC051642	319	1-462
HNGEA34	67	815678	AC068137	320	1-1100
HNGKT41	69	836061	AC008581	321	1-1099
HNGNO53	70	836063	AC023387	322	1-869
HNGNO53	70	836063	AL355500	323	1-851
HNHCT47	71	634691	AC027793	324	1-147
HNHCT47	71	634691	AC022107	325	1-111
HNHCT47	71	634691	AP001271	326	1-610
HNHCT47	71	634691	AP000487	327	1-610
HNHCT47	71	634691	AP000405	328	1-612
HNHCT47	71	634691	AP001271	329	1-375
HNHCT47	71	634691	AP000487	330	1-36 434-873
HNHCT47	71	634691	AP000405	331	1-375
HORBS82	73	638293	AL034419	332	1-1798
HORBS82	73	638293	AL034419	333	1-1186
HOUDE92	74	580866	AC005865	334	1-173 553-629 1941-2042

					2757-2891 3294-3378 4606-5498 5550-8125
HPCAL26	77	762822	AP000654	335	1-4150
HPEBA84	78	753957	AL357372	336	1-1238
HPEBA84	78	753957	AL161799	337	1-1112
HPEBA84	78	753957	AL357372	338	1-294
HPEBA84	78	753957	AL357372	339	1-140
HPEBA84	78	753957	AL161799	340	1-294
HSAVA08	79	580870	AC009030	341	1-1052
HSAVA08	79	580870	AC009030	342	1-431
HSAX04	80	812178	AL049824	343	1-110 1211-1257 1740-1878 3062-3144 3668-3772 4775-5175 5220-5345 7001-7384 8361-8657 8747-8937 9876-9980 12753-12901 13131-13891 14272-14726 14851-16619 16683-17910 18078-18367
HSAX04	80	812178	AL354888	344	1-47 1277-1376 2477-2523 3006-3144 4326-4408 4932-5036 6039-6439 6484-6609 8265-8648 9625-9921 10011-10216 11141-11245 14019-14167 14397-15157 15538-15992 16117-17885 17949-19176 19344-19633
HSAX04	80	812178	AL354888	345	1-314
HSAX04	80	812178	AL354888	346	1-599
HSKDR27	81	580874	AC008742	347	1-50 1016-1321 1979-2220

					2313-3310
HSKDR27	81	580874	AC008742	348	1-495
HSQBF66	82	560726	AC011878	349	1-117 4124-5072 5221-5252
HSRFD18	83	840771	AL096819	350	1-3975
HSRFD18	83	840771	AL096819	351	1-304
HT3BF49	85	838620	AL355304	352	1-2144
HT3BF49	85	838620	AL355307	353	1-2144
HT3BF49	85	838620	AL355304	354	1-517
HT3BF49	85	838620	AL355307	355	1-517
HTEHU59	87	840385	AP001003	356	1-3207
HTEHU59	87	840385	AP001557	357	1-3206
HTEHU59	87	840385	AP001156	358	1-3207
HTEHU59	87	840385	AP001003	359	1-863
HTEHU59	87	840385	AP001003	360	1-1399 1504-1948 1956-2672 2761-2905 3007-3135 3290-3445 3537-3653 3746-3913 4010-4131 4251-4428
HTEHU59	87	840385	AP001557	361	1-863
HTEHU59	87	840385	AP001557	362	1-1395 1500-1944 1952-2667 2757-2900 3002-3130 3285-3439
HTEHU59	87	840385	AP001156	363	1-1396 1502-1945 1953-2668
HTEHU59	87	840385	AP001156	364	1-863
HTTEE41	95	840950	AC018921	365	1-92 318-578 837-912 1091-1249 1321-1387 1862-2192 2485-2579 2708-2831 3685-4257 4547-5127 5811-6037 6562-7076 7541-7678 8069-8191 10100-10207 11102-11688

					11721-11847 12201-12335 12532-12641 12888-12991 13027-13546 13637-16146
HTTEE41	95	840950	AC018921	366	1-100
HTXJD85	96	840391	AC078797	367	1-1239
HTXJD85	96	840391	AC078797	368	1-2296 2428-2719
HTXJD85	96	840391	AC078797	369	1-224
HWHPB78	99	740778	AL157945	370	1-300 364-790 1344-1519 1584-1709 2403-2580 4780-4968 5485-5559 5960-6128 6243-6955 7258-7317 9073-9145 9404-9544 10342-10513 10746-11354 12004-12578 12863-13087 13224-13382 13993-14047 14319-14444 14753-14878 15465-15713 16007-16123 17413-17740 17817-18127 18231-18634 18771-18881 19945-20231 21024-21169 23112-23363 23692-24413
HWHPB78	99	740778	AC026283	371	1-292 353-776 1340-1506 1568-1696 2408-2534 4767-4955 5472-5546 5957-6293 6373-7085 7386-7445 9201-9273

					9532-9672 10470-10641 10873-11481 12131-12705 12990-13214 13351-13509 14119-14173 14445-14570 14879-15004 15604-15844 16133-16253 17540-17867 17944-18254 18356-18755 18892-19002 20066-20352 21146-21308 23235-23486 23813-24533
HWHPB78	99	740778	AL157945	372	1-490
HWHPB78	99	740778	AC026283	373	1-318
HWLBO67	100	834315	AC011739	374	1-517
HWLBO67	100	834315	AC011739	375	1-586 3120-3867 4726-4866 6044-6395 6686-7156 11614-12016 18205-18501
HWLBO67	100	834315	AC011739	376	1-202
HE2CA60	103	888705	AC005921	377	1-74 276-1076 1472-2160 3055-3389 3769-3898 4143-4288 4322-4697 4699-4772 6745-6851 7692-9044 9581-9743 13540-17646 1-74 276-1076 1472-2160 3055-3389 3769-3898 4143-4288 4322-4697 4699-4772 6745-6851 7692-9044

					9581-9743 13540-17646
HE2CA60	103	888705	AC005921	378	1-1466 1-1466
HEQBJ01	106	876546	AC009079	379	1-198 1263-1467 2271-2369 5035-5870 5888-6174 6249-6472 7148-11343 1-198 1263-1467 2271-2369 5035-5870 5888-6174 6249-6472 7148-11343
HEQBJ01	106	876546	AC009079	380	1-173 916-970 1-173 916-970

Tables 1D: The polynucleotides or polypeptides, or agonists or antagonists of the present invention can be used in assays to test for one or more biological activities. If these polynucleotides and polypeptides do exhibit activity in a particular assay, it is likely that these molecules may be involved in the diseases associated with the biological activity. Thus, the polynucleotides or polypeptides, or agonists or antagonists could be used to treat the associated disease.

The present invention encompasses methods of detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating a disease or disorder. In preferred embodiments, the present invention encompasses a method of treating an allergic and/or asthmatic disease or disorder comprising administering to a patient in which such detection, treatment, prevention, and/or amelioration is desired a protein, nucleic acid, or antibody of the invention (or fragment or variant thereof) in an amount effective to detect, prevent, diagnose, prognosticate, treat, and/or ameliorate the allergic and/or asthmatic disease or disorder.

In another embodiment, the present invention also encompasses methods of detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating an allergic and/or asthmatic disease or disorder; comprising administering to a patient combinations of the proteins, nucleic acids, or antibodies of the invention (or fragments or variants thereof), sharing similar indications

as shown in the corresponding rows in Column 3 of Table 1D.

Table 1D provides information related to biological activities for polynucleotides and polypeptides of the invention (including antibodies, agonists, and/or antagonists thereof). Table 1D also provides information related to assays which may be used to test polynucleotides and polypeptides of the invention (including antibodies, agonists, and/or antagonists thereof) for the corresponding biological activities. The first column ("Gene No.") provides the gene number in the application for each clone identifier. The second column ("cDNA Clone ID:") provides the unique clone identifier for each clone as previously described and indicated in Table 1A through Table 1D. The third column ("AA SEQ ID NO:Y") indicates the Sequence Listing SEQ ID Number for polypeptide sequences encoded by the corresponding cDNA clones (also as indicated in Tables 1A, Table 1B, and Table 2). The fourth column ("Biological Activity") indicates a biological activity corresponding to the indicated polypeptides (or polynucleotides encoding said polypeptides). The fifth column ("Exemplary Activity Assay") further describes the corresponding biological activity and also provides information pertaining to the various types of assays that may be performed to test, demonstrate, or quantify the corresponding biological activity.

Table 1D describes the use of, inter alia, FMAT technology for testing or demonstrating various biological activities. Fluorometric microvolume assay technology (FMAT) is a fluorescence-based system that provides a means to perform nonradioactive cell- and bead-based assays to detect activation of cell signal transduction pathways. This technology was designed specifically for ligand binding and immunological assays. Using this technology, fluorescent cells or beads at the bottom of the well are detected as localized areas of concentrated fluorescence using a data processing system. Unbound fluorophore comprising the background signal is ignored, allowing for a wide variety of homogeneous assays. FMAT technology may be used for peptide ligand binding assays, immunofluorescence, apoptosis, cytotoxicity, and bead-based immunocapture assays. *See*, Miraglia S et. al., "Homogeneous cell and bead based assays for highthroughput screening using fluorometric microvolume assay technology," *Journal of Biomolecular Screening*; 4:193-204 (1999). In particular, FMAT technology may be used to test, confirm, and/or identify the ability of polypeptides (including polypeptide fragments and variants) to activate signal transduction pathways. For example, FMAT technology may be used to test, confirm, and/or identify the ability of polypeptides to upregulate production of immunomodulatory proteins (such as, for example, interleukins, GM-CSF, Rantes, and Tumor Necrosis factors, as well as other cellular regulators (e.g. insulin)).

Table 1D also describes the use of kinase assays for testing, demonstrating, or quantifying biological activity. In this regard, the phosphorylation and de-phosphorylation

of specific amino acid residues (e.g. Tyrosine, Serine, Threonine) on cell-signal transduction proteins provides a fast, reversible means for activation and de-activation of cellular signal transduction pathways. Moreover, cell signal transduction via phosphorylation/de-phosphorylation is crucial to the regulation of a wide variety of cellular processes (e.g. proliferation, differentiation, migration, apoptosis, etc.). Accordingly, kinase assays provide a powerful tool useful for testing, confirming, and/or identifying polypeptides (including polypeptide fragments and variants) that mediate cell signal transduction events via protein phosphorylation. See e.g., Forrer, P., Tamaskovic R., and Jaussi, R. "Enzyme-Linked Immunosorbent Assay for Measurement of JNK, ERK, and p38 Kinase Activities" *Biol. Chem.* 379(8-9): 1101-1110 (1998).

Table 1D

Gene No.	cDNA Clone ID	AA SEQ ID NO: Y	Biological Activity	Exemplary Activity Assay	Preferred Indication
1	HACBT91	118	Production of IL-10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics;	Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.

				<p>88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.</p>	
2	HADDE71	119	<p>Activation of transcription through cAMP response element (CRE) in pre-adipocytes.</p>	<p>Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription</p>	<p>A highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. An additional highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g.,</p>

			<p>factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated</p>	<p>diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyposmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders</p>
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				by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.	as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). Additional highly preferred indications are complications associated with insulin resistance.
3	HADDJ13	120	Activation of transcription through cAMP response element (CRE) in pre-adipocytes.	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions.	A highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. An additional highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy

			<p>For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are</p>	<p>and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract</p>
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				publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.	and skin), carpal tunnel syndrome and Dupuytren's contracture). Additional highly preferred indications are complications associated with insulin resistance.
3	HADDJ13	120	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3	Highly preferred indications include allergy, asthma, and rhinitis. Additional preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications also include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic

			<p>transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available</p>	<p>lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary tract cancers and/or as described below under "Hyperproliferative Disorders"). Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes</p>
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				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	mellitus, endocarditis, meningitis, and Lyme Disease.
3	HADDJ13	120	Activation of transcription through NFAT response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory	Highly preferred indications include allergy, asthma, and rhinitis. Additional preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications also include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below).

				<p>functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature</p>	<p>Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary tract cancers and/or as described below under "Hyperproliferative Disorders"). Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
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				human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	
3	HADDJ13	120	Activation of transcription through NFAT response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol	Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include

				<p>216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.</p>	<p>neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis,</p>
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					meningitis, Lyme Disease, asthma and allergy.
3	HADDJ13	120	<p>Activation of transcription through GAS response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988);</p>	<p>Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma (e.g., T cell lymphoma, Burkitt's lymphoma, non-Hodgkins lymphoma, Hodgkin's disease), melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response,</p>

				<p>Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the SUPT cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).</p>	<p>and suppressing a T cell-mediated immune response. Additional preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., viral infections, tuberculosis, infections associated with chronic granulomatous disease and malignant osteoporosis, and/or an infectious disease as described below under "Infectious Disease"). An additional preferred indication is idiopathic pulmonary fibrosis. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs</p>
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					and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, and asthma and allergy.
4	HADMA77	121	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	<p>Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature</p>	<p>Highly preferred indications include asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below). Highly preferred indications also include boosting or inhibiting immune cell proliferation. Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an eosinophil-</p>

				<p>410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000);</p>	mediated immune response, and suppressing an eosinophil-mediated immune response.
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				<p>Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.</p>	
5	HADMB15	122	<p>Regulation of transcription via DMEF1 response element in adipocytes and pre-adipocytes</p>	<p>Assays for the regulation of transcription through the DMEF1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the DMEF1 response element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate</p> <p>A highly preferred indication is diabetes mellitus. Additional highly preferred indications include complications associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic</p>	

			<p>insulin production. The DMEF1 response element is present in the GLUT4 promoter and binds to MEF2 transcription factor and another transcription factor that is required for insulin regulation of Glut4 expression in skeletal muscle. GLUT4 is the primary insulin-responsive glucose transporter in fat and muscle tissue. Exemplary assays that may be used or routinely modified to test for DMEF1 response element activity (in adipocytes and pre-adipocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Thai, M.V., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 275(21):16323-8 (2000); Liu, M.L., et al., J Biol Chem, 269(45):28514-21 (1994); "Identification of a 30-base pair regulatory element and novel DNA binding protein that regulates the human GLUT4 promoter in transgenic mice", J Biol Chem. 2000 Aug 4;275(31):23666-73; Berger, et al., Gene 66:1-10 (1988); and,</p>	<p>neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively,</p>
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				<p>Cullen, B., et al., Methods in Enzymol. 216:362–368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Adipocytes and pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include the mouse 3T3-L1 cell line which is an adherent mouse preadipocyte cell line. Mouse 3T3-L1 cells are a continuous substrain of 3T3 fibroblasts developed through clonal isolation. These cells undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation culture conditions.</p>	<p>weight gain. Additional highly preferred indications are complications associated with insulin resistance.</p>
5	HADMB15	122	<p>Regulation of apoptosis of immune cells (such as mast cells).</p>	<p>Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate caspase protease-mediated apoptosis in</p>	<p>Preferred embodiments of the invention include using polypeptides of the invention (or antibodies, agonists, or antagonists thereof) in detection, diagnosis, prevention, and/or treatment of asthma, allergy, hypersensitivity and inflammation.</p>

				<p>immune cells (such as, for example, in mast cells). Mast cells are found in connective and mucosal tissues throughout the body, and their activation via immunoglobulin E-antigen, promoted by T helper cell type 2 cytokines, is an important component of allergic disease. Dysregulation of mast cell apoptosis may play a role in allergic disease and mast cell tumor survival. Exemplary assays for caspase apoptosis that may be used or routinely modified to test caspase apoptosis activity induced by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Masuda A, et al., J Biol Chem, 276(28):26107-26113 (2001); Yeatman CF 2nd, et al., J Exp Med, 192(8):1093-1103 (2000); Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its</p>	
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5	HADMB15	122	Activation of Natural Killer Cell ERK Signaling Pathway.	<p>entirety. Immune cells that may be used according to these assays are publicly available (e.g., through commercial sources). Exemplary immune cells that may be used according to these assays include mast cells such as the HMC human mast cell line.</p> <p>Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal transduction that regulate cell proliferation or differentiation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-</p>	<p>A highly preferred embodiment of the invention includes a method for stimulating natural killer cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting natural killer cell proliferation. A highly preferred embodiment of the invention includes a method for stimulating natural killer cell differentiation. An alternative highly preferred embodiment of the invention includes a method for inhibiting natural killer cell differentiation. Highly preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as described below under "Immune</p>
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				<p>9):1101-1110 (1998); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety.</p> <p>Natural killer cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary natural killer cells that may be used according to these assays include the human natural killer cell lines (for example, NK-YT cells which have cytolytic and cytotoxic activity) or primary NK cells.</p>	<p>Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), immune disorders (e.g., as described below under "Immune Activity") and infections (e.g., as described below under "Infectious Disease").</p> <p>Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders").</p> <p>Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional highly preferred indications include inflammation and inflammatory disorders.</p> <p>Highly preferred indications also include cancers such as, kidney, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, urinary cancer, lymphoma and leukemias. Other preferred indications include benign</p>
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6	HAGBQ12	123	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to	dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Other highly preferred indications include, pancytopenia, leukopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), arthritis, asthma, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, psoriasis, immune reactions to transplanted organs and tissues, endocarditis, meningitis, Lyme Disease, and allergies.
				Highly preferred indications include asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below),	

				<p>test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate</p>	<p>immunodeficiencies (e.g., as described below). Highly preferred indications also include boosting or inhibiting immune cell proliferation. Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.</p>
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				<p>signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.</p>	
				<p>IFNgamma FMAT. IFNg plays a central role in the immune</p>	<p>A highly preferred embodiment of the invention</p>
6	HAGBQ12	123	Production of IFNgamma using a		

			<p>T cells</p>	<p>system and is considered to be a proinflammatory cytokine. IFNg promotes TH1 and inhibits TH2 differentiation; promotes IgG2a and inhibits IgE secretion; induces macrophage activation; and increases MHC expression. Assays for immunomodulatory proteins produced by T cells and NK cells that regulate a variety of inflammatory activities and inhibit TH2 helper cell functions are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, regulate inflammatory activities, modulate TH2 helper cell function, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as Interferon gamma (IFNg), and the activation of T cells. Such assays that may be used or routinely modified to test</p>	<p>includes a method for stimulating the production of IFNg. An alternative highly preferred embodiment of the invention includes a method for inhibiting the production of IFNg. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., viral infections, tuberculosis, infections associated with chronic granulomatous disease and malignant osteoporosis, and/or as described below under "Infectious Disease"). Highly preferred indications include autoimmune disease (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiency (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory</p>
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				<p>immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Gonzalez et al., J Clin Lab Anal 8(5):225-233 (1995); Billiau et al., Ann NY Acad Sci 856:22-32 (1998); Boehm et al., Annu Rev Immunol 15:749-795 (1997), and Rheumatology (Oxford) 38(3):214-20 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T Cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance</p>	<p>disorders. Additional preferred indications include idiopathic pulmonary fibrosis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis,</p>
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					responsiveness to immunomodulatory factors.	suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.
7	HAGCC87	124	Production of IL-13 and activation of T-cells.	Assays for production of IL-13 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-13 and/or activation of T-cells. Exemplary assays for IL-13 production that may be used or routinely modified to test activity of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) include, for example, assays such as disclosed and/or cited in: Grunig, G, et al., "Requirement for IL-13 independently of IL-4 in Experimental asthma" Science;282: 2261-2263 (1998),	Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.	

				and Wills-Karp M, et al., "Interleukin-13: central mediator of allergic asthma" Science; 282: 2258-2261 (1998); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL13, a Th2 type cytokine, is a potent stimulus for mucus production, airway hyper-responsiveness and allergic asthma. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
8	HAGHN57	125	Activation of transcription through NFAT response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely	Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders").

			<p>modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by</p>	<p>Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative</p>
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				reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.	disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.
8	HAGHN57	125	Production of IL-10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or	Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus

				<p>activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis</p>	<p>erythematosis, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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				of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
9	HAGHR18	126	Production of IL-10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed	Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.

				therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
10	HAQA192	127	Regulation of proliferation and/or differentiation in immune cells (such as mast cells).	Kinase assays, for example an Elk-1 kinase assay for ERK signal transduction that regulates cell proliferation or differentiation, are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies	Preferred embodiments of the invention include using polypeptides of the invention (or antibodies, agonists, or antagonists thereof) in detection, diagnosis, prevention, and/or treatment of asthma, allergy, hypersensitivity and inflammation.

				<p>and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Ali H, et al., J Immunol, 165(12):7215-7223 (2000); Tam SY, et al., Blood, 90(5):1807-1820 (1997); Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Berra et al., Biochem Pharmacol 60(8):1171-1178 (2000); Gupta et al., Exp Cell Res 247(2):495-504 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary immune cells that may be used according to these assays include human mast cells such as the HMC-1 cell line.</p>
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11	HAQBG57	128	<p>Production of IL-10 and activation of T-cells.</p>	<p>Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to</p>	<p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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				these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
12	HAQCE11	129	Production of IL-5	IL-5 FMAT. Assays for immunomodulatory proteins secreted by TH2 cells, mast cells, basophils, and eosinophils that stimulate eosinophil function and B cell Ig production and promote polarization of CD4+ cells into TH2 cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, stimulate	<p>A highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-5 production.</p> <p>An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-5 production. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) immunoglobulin production. An alternative highly preferred embodiment of the invention includes a method for inhibiting</p>

			<p>immune cell function, modulate B cell Ig production, modulate immune cell polarization, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-5, and the stimulation of eosinophil function and B cell Ig production. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Ohshima et al., Blood 92(9):3338-3345 (1998); Jung et al., Eur J Immunol 25(8):2413-2416 (1995); Mori et al., J Allergy Clin Immunol 106(1 Pt 2):558-564 (2000); and Koning et al., Cytokine 9(6):427-436 (1997), the contents of each of which are herein incorporated</p>	<p>(e.g., decreasing) immunoglobulin production. A highly preferred indication includes allergy. A highly preferred indication includes asthma. A highly preferred indication includes rhinitis. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms</p>
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				<p>by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.</p>	<p>and cancers, such as, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
13	HBAGD86	130	<p>Regulation of transcription via DMEF1 response element in adipocytes and pre-</p>	<p>Assays for the regulation of transcription through the DMEF1 response element are well-known in the art and may be used or routinely modified to</p>	<p>A highly preferred indication is diabetes mellitus. Additional highly preferred indications include complications associated with diabetes (e.g.,</p>

		adipocytes	<p>assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the DMEF1 response element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insulin production. The DMEF1 response element is present in the GLUT4 promoter and binds to MEF2 transcription factor and another transcription factor that is required for insulin regulation of Glut4 expression in skeletal muscle. GLUT4 is the primary insulin-responsive glucose transporter in fat and muscle tissue. Exemplary assays that may be used or routinely modified to test for DMEF1 response element activity (in adipocytes and pre-adipocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Thai, M.V., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 275(21):16323-8 (2000); Liu, M.L., et al., J Biol Chem,</p>	<p>diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hypermolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders</p>
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				<p>269(45):28514-21 (1994); "Identification of a 30-base pair regulatory element and novel DNA binding protein that regulates the human GLUT4 promoter in transgenic mice", J Biol Chem. 2000 Aug 4;275(31):23666-73; Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Adipocytes and pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include the mouse 3T3-L1 cell line which is an adherent mouse preadipocyte cell line. Mouse 3T3-L1 cells are a continuous substrain of 3T3 fibroblasts developed through clonal isolation. These cells undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation culture conditions.</p>	<p>as described in the "Infectious Diseases" section below, especially of the urinary tract and skin). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance.</p>
	HBAGD86	130	Activation of	Assays for the activation of	A highly preferred indication is

13	transcription through cAMP response element (CRE) in pre-adipocytes.	transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene	obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. An additional highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hypermolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular
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13	HBAGD86	130	Activation of transcription through serum response element in pre-	<p>66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.</p> <p>Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may</p>	<p>Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). Additional highly preferred indications are complications associated with insulin resistance.</p>	<p>A highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred</p>
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			adipocytes.	<p>be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may</p>	<p>indications include weight loss or alternatively, weight gain. An additional highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyposmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the</p>
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				be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.	"Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below). Additional highly preferred indications are complications associated with insulin resistance.
13	HBAGD86	130	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast	Highly preferred indications include allergy, asthma, and rhinitis. Additional preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications also include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described

			<p>cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells</p>	<p>below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary tract cancers and/or as described below under "Hyperproliferative Disorders"). Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
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13	HBAGD86	130	Activation of transcription through NFAT response element in immune cells (such as mast cells).	that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	Highly preferred indications include allergy, asthma, and rhinitis. Additional preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications also include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g.,
				This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT	

			<p>response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a</p>	<p>leukemia, lymphoma, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary tract cancers and/or as described below under "Hyperproliferative Disorders"). Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
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				patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	
13	HBAGD86	130	Activation of transcription through NFkB response element in immune cells (such as mast cells).	<p>This reporter assay measures activation of the NFkB signaling pathway in HMC-1 human mast cell line. Activation of NFkB in mast cells has been linked to production of certain cytokines, such as IL-6 and IL-9. Assays for the activation of transcription through the NFkB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFkB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFkB response element that may be used or routinely modified to test NFkB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of</p>	<p>Highly preferred indication includes allergy, asthma, and rhinitis. Additional highly preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications include immunological and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"). Preferred indications also include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred</p>

				<p>the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Stassen et al, J Immunol 166(7):4391-8 (2001); and Marquardt and Walker, J Allergy Clin Immunol 105(3):500-5 (2000), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.</p>	<p>indications include neoplasms and cancer, such as, for example, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, urinary tract cancers and as described below under "Hyperproliferative Disorders".</p>
13	HBAGD86	130	<p>Activation of transcription through NFkB response element in immune cells (such as</p>	<p>This reporter assay measures activation of the NFkB signaling pathway in Ku812 human basophil cell line. Assays for the activation of transcription</p>	<p>Highly preferred indication includes allergy, asthma, and rhinitis. Additional highly preferred indications include infection (e.g., an infectious</p>

			<p>basophils).</p>	<p>through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Marone et al, Int Arch Allergy Immunol 114(3):207-17 (1997), the contents of each of which are herein incorporated by reference in its entirety. Basophils that may be used according to these assays are publicly available</p>	<p>disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications include immunological and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"). Preferred indications also include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancer, such as, for example, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, urinary tract cancers and as described below under "Hyperproliferative Disorders".</p>
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				(e.g., through the ATCC). Exemplary human basophil cell lines that may be used according to these assays include Ku812, originally established from a patient with chronic myelogenous leukemia. It is an immature prebasophilic cell line that can be induced to differentiate into mature basophils.	
13	HBAGD86	130	Activation of transcription through NFAT response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies	Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. An additional highly

			<p>and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.</p>	<p>preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis,</p>
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13	HBAGD86	130	<p>Activation of transcription through GAS response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10</p>	<p>suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.</p> <p>Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma (e.g., T cell lymphoma, Burkitt's lymphoma, non-Hodgkins lymphoma, Hodgkin's disease), melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus,</p>
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				<p>(1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the SUPT cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).</p>	<p>multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., viral infections, tuberculosis, infections associated with chronic granulomatous disease and malignant osteoporosis, and/or an infectious disease as described below under "Infectious Disease"). An additional preferred indication is idiopathic pulmonary fibrosis. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, arthritis, AIDS, granulomatous disease,</p>
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14	HGBC29	131	Protection from Endothelial Cell Apoptosis.	<p>Caspase Apoptosis Rescue. Assays for caspase apoptosis rescue are well known in the art and may be used or routinely modified to assess the ability of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to inhibit caspase protease-mediated apoptosis. Exemplary assays for caspase apoptosis that may be used or routinely modified to test caspase apoptosis rescue of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Romeo et al., Cardiovasc Res 45(3): 788-794 (2000); Messmer et al., Br J Pharmacol 127(7): 1633-1640</p>	<p>inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, and asthma and allergy.</p> <p>A highly preferred embodiment of the invention includes a method for stimulating endothelial cell growth. An alternative highly preferred embodiment of the invention includes a method for inhibiting endothelial cell growth. A highly preferred embodiment of the invention includes a method for stimulating endothelial cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting endothelial cell proliferation. A highly preferred embodiment of the invention includes a method for stimulating endothelial cell growth. An alternative highly preferred embodiment of the</p>
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				<p>(1999); and J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays are publicly available (e.g., through commercial sources). Exemplary endothelial cells that may be used according to these assays include bovine aortic endothelial cells (bAEC), which are an example of endothelial cells which line blood vessels and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.</p>	<p>invention includes a method for inhibiting endothelial cell growth. A highly preferred embodiment of the invention includes a method for stimulating apoptosis of endothelial cells. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., decreasing) apoptosis of endothelial cells. A highly preferred embodiment of the invention includes a method for stimulating angiogenesis. An alternative highly preferred embodiment of the invention includes a method for inhibiting angiogenesis. A highly preferred embodiment of the invention includes a method for reducing cardiac hypertrophy. An alternative highly preferred embodiment of the invention includes a method for inducing cardiac hypertrophy. Highly preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), and disorders of the cardiovascular system (e.g., heart disease, congestive heart</p>
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				<p>failure, hypertension, aortic stenosis, cardiomyopathy, valvular regurgitation, left ventricular dysfunction, atherosclerosis and atherosclerotic vascular disease, diabetic nephropathy, intracardiac shunt, cardiac hypertrophy, myocardial infarction, chronic hemodynamic overload, and/or as described below under “Cardiovascular Disorders”).</p> <p>Highly preferred indications include cardiovascular, endothelial and/or angiogenic disorders (e.g., systemic disorders that affect vessels such as diabetes mellitus, as well as diseases of the vessels themselves, such as of the arteries, capillaries, veins and/or lymphatics). Highly preferred are indications that stimulate angiogenesis and/or cardiovascularization. Highly preferred are indications that inhibit angiogenesis and/or cardiovascularization.</p> <p>Highly preferred indications include antiangiogenic activity to treat solid tumors, leukemias, and Kaposi's sarcoma, and</p>
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				<p>retinal disorders. Highly preferred indications include neoplasms and cancer, such as, Kaposi's sarcoma, hemangioma (capillary and cavernous), glomus tumors, telangiectasia, bacillary angiomatosis, hemangioendothelioma, angiosarcoma, haemangiopericytoma, lymphangioma, lymphangiosarcoma. Highly preferred indications also include cancers such as, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Highly preferred indications also include arterial disease, such as, atherosclerosis, hypertension, coronary artery disease, inflammatory vasculitides, Reynaud's disease and Reynaud's phenomenon, aneurysms, restenosis; venous and lymphatic disorders such as thrombophlebitis, lymphangitis, and lymphedema; and other</p>
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					<p>vascular disorders such as peripheral vascular disease, and cancer. Highly preferred indications also include trauma such as wounds, burns, and injured tissue (e.g., vascular injury such as, injury resulting from balloon angioplasty, and atherosclerotic lesions), implant fixation, scarring, ischemia reperfusion injury, rheumatoid arthritis, cerebrovascular disease, renal diseases such as acute renal failure, and osteoporosis. Additional highly preferred indications include stroke, graft rejection, diabetic or other retinopathies, thrombotic and coagulative disorders, vasculitis, lymph angiogenesis, sexual disorders, age-related macular degeneration, and treatment /prevention of endometriosis and related conditions. Additional highly preferred indications include fibromas, heart disease, cardiac arrest, heart valve disease, and vascular disease. Preferred indications include blood disorders (e.g., as described below under “Immune</p>
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					Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional preferred indications include inflammation and inflammatory disorders (such as acute and chronic inflammatory diseases, e.g., inflammatory bowel disease and Crohn's disease), and pain management.
14	HBGBC29	131	Inhibition of squalene synthetase gene transcription.	Reporter Assay: construct contains regulatory and coding sequence of squalene synthetase, the first specific enzyme in the cholesterol biosynthetic pathway. See Jiang, et al., J. Biol. Chem. 268:12818-12824(1993), the contents of which are herein incorporated by reference in its entirety. Cells were treated with SID supernatants, and SEAP activity was measured after 72 hours. HepG2 is a human hepatocellular carcinoma cell	

14	HBGBC29	131	Production of IL-10 and activation of T-cells.	<p>line (ATCC HB-8065). See Knowles et al., Science. 209:497-9 (1980), the contents of which are herein incorporated by reference in its entirety.</p> <p>Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma"</p>	<p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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15	HBJAB02	132		<p>Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.</p> <p>Assays for measuring calcium flux are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mobilize calcium. Cells normally have very low concentrations of cytosolic</p>	<p>Preferred embodiments of the invention include using polypeptides of the invention (or antibodies, agonists, or antagonists thereof) in detection, diagnosis, prevention, and/or treatment of Bone and Cartilage Diseases, including but not limited to Arthritis, Cartilage repair, Bone Repair,</p>
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				<p>calcium compared to much higher extracellular calcium. Extracellular factors can cause an influx of calcium, leading to activation of calcium responsive signaling pathways and alterations in cell functions. Exemplary assays that may be used or routinely modified to measure calcium flux in chondrocytes include assays disclosed in: Asada S, et al., <i>Inflamm Res</i>, 50(1):19-23 (2001); Schwartz Z, et al., <i>J Bone Miner Res</i>, 6(7):709-718 (1991); Iannotti JP, et al., <i>J Bone Joint Surg Am</i>, 67(1):113-120 (1985); Sullivan E., et al., <i>Methods Mol Biol</i> 1999; 114:125-133 (1999), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include bovine chondrocytes.</p>	<p>Osteoporosis, and related tumors including chondrosarcomas, chondroblastomas, and chondromas.</p>
15	HBJAB02	132	Activation of JNK Signaling Pathway	<p>Kinase assay. JNK kinase assays for signal transduction</p>	<p>Highly preferred indications include asthma, allergy,</p>

			<p>in immune cells (such as eosinophils).</p>	<p>that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays</p>	<p>hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below). Highly preferred indications also include boosting or inhibiting immune cell proliferation. Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.</p>
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				<p>include eosinophils.</p> <p>Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in</p>	
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				bronchial asthma is associated with enhanced phosphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.	
16	HBMUH74	133	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110	Highly preferred indications include asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below). Highly preferred indications also include boosting or inhibiting immune cell proliferation. Preferred indications include neoplastic diseases (e.g.,

				<p>(1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation</p>	<p>leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.</p>
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				<p>of c-Jun NH2-terminal kinase and p38 mitogen-activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.</p>	
16	HBMUH74	133	<p>Regulation of transcription of Malic Enzyme in adipocytes</p>	<p>Assays for the regulation of transcription of Malic Enzyme are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate</p>	<p>A highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and</p>

			transcription of Malic Enzyme, a key enzyme in lipogenesis. Malic enzyme is involved in lipogenesis and its expression is stimulated by insulin. ME promoter contains two direct repeat (DR1)-like elements MEp and MEd identified as putative PPAR response elements. ME promoter may also respond to API and other transcription factors. Exemplary assays that may be used or routinely modified to test for regulation of transcription of Malic Enzyme (in adipocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Streeter, R.S., et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol, 8(10):1361-9 (1994); Barroso, I., et al., J Biol Chem, 274(25):17997-8004 (1999); Ijpenberg, A., et al., J Biol Chem, 272(32):20108-20117 (1997); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992),	disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hypermolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel
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				<p>the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays includes the H4IIE rat liver hepatoma cell line.</p>	<p>syndrome and Dupuytren's contracture). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance.</p>
17	HBNAX40	134	<p>Activation of transcription through GATA-3 response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of genes important for Th2 immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of</p>	<p>A highly preferred indication includes allergy. A highly preferred indication includes asthma. A highly preferred indication includes rhinitis. Additional highly preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g.,</p>

				<p>polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the HT2 cell line, which is a suspension culture of IL-2 dependent T cells that also respond to IL-4.</p>	<p>rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancer, such as, for example, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel</p>
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					<p>disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
18	HBXCX15	135	<p>Regulation of transcription via DMEF1 response element in adipocytes and pre-adipocytes</p>	<p>Assays for the regulation of transcription through the DMEF1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the DMEF1 response element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insulin production. The DMEF1 response element is present in the GLUT4 promoter and binds to MEF2 transcription factor and another transcription factor that is required for insulin regulation of Glut4 expression in skeletal muscle. GLUT4 is the primary insulin-responsive glucose transporter in fat and</p>	<p>A highly preferred indication is diabetes mellitus. Additional highly preferred indications include complications associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis,</p>

			<p>muscle tissue. Exemplary assays that may be used or routinely modified to test for DMEF1 response element activity (in adipocytes and pre-adipocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Thai, M. V., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 275(21):16323-8 (2000); Liu, M.L., et al., J Biol Chem, 269(45):28514-21 (1994); "Identification of a 30-base pair regulatory element and novel DNA binding protein that regulates the human GLUT4 promoter in transgenic mice", J Biol Chem. 2000 Aug 4;275(31):23666-73; Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Adipocytes and pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary</p>	<p>microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance.</p>
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				cells that may be used according to these assays include the mouse 3T3-L1 cell line which is an adherent mouse preadipocyte cell line. Mouse 3T3-L1 cells are a continuous substrain of 3T3 fibroblasts developed through clonal isolation. These cells undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation culture conditions.	
18	HBXCX15	135	Activation of transcription through cAMP response element (CRE) in pre-adipocytes.	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in	A highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. An additional highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic

				<p>differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells.</p>	<p>neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyposmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). Additional highly preferred indications are complications associated with insulin resistance.</p>
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18	HBXCX15	135	<p>Activation of transcription through serum response element in pre-adipocytes.</p>	<p>3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.</p> <p>Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm,</p>	<p>A highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. An additional highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic</p>
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				<p>Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.</p>	<p>neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hypermolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below). Additional highly preferred indications are complications associated with insulin resistance.</p>
18	HBXCX15	135	<p>Activation of transcription through GATA-3 response element in immune cells (such as mast</p>	<p>This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has</p>	<p>Highly preferred indications include allergy, asthma, and rhinitis. Additional preferred indications include infection (e.g., an infectious disease as</p>

			cells).	<p>been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp</p>	<p>described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications also include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary tract cancers and/or as described below under "Hyperproliferative Disorders"). Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia,</p>
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				<p>Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.</p>	<p>leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
18	HBXCX15	135	<p>Activation of transcription through NFAT response element in immune cells (such as mast cells).</p>	<p>This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the</p>	<p>Highly preferred indications include allergy, asthma, and rhinitis. Additional preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications</p>

				<p>Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995),</p>	<p>also include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary tract cancers and/or as described below under "Hyperproliferative Disorders"). Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma,</p>
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				<p>and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.</p>	<p>arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
18	HBXCX15	135	<p>Activation of transcription through NFAT response element in immune cells (such as natural killer cells).</p>	<p>Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory</p>	<p>Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T</p>

				<p>functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays include the NK-YT cell line, which is a human</p>	<p>cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic</p>
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				natural killer cell line with cytolytic and cytotoxic activity.	anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.
18	HBXCX15	135	Activation of serum response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE	A preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) TNF alpha production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) TNF alpha production. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis,

				<p>activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.</p>	<p>systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders, and treating joint damage in patients with rheumatoid arthritis. An additional highly preferred indication is sepsis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Additionally, highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, glioma (e.g., malignant glioma), solid tumors, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative</p>
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18	HBXCX15	135	<p>Activation of transcription through GAS response element in immune cells (such as T-cells).</p> <p>Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to</p>	<p>disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, cardiac reperfusion injury, and asthma and allergy. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").</p> <p>Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders").</p> <p>Highly preferred indications</p>
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			<p>assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Hentinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the SUPT cell line, that may be used</p>	<p>include neoplasms and cancers, such as, for example, leukemia, lymphoma (e.g., T cell lymphoma, Burkitt's lymphoma, non-Hodgkins lymphoma, Hodgkin's disease), melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related</p>
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				according to these assays are publicly available (e.g., through the ATCC).	Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., viral infections, tuberculosis, infections associated with chronic granulomatous disease and malignant osteoporosis, and/or an infectious disease as described below under "Infectious Disease"). An additional preferred indication is idiopathic pulmonary fibrosis. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, and asthma and allergy.
19	HCDBO32	136	Production of IL-10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may	Highly preferred indications include allergy and asthma. Additional highly preferred

				<p>be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of</p>	<p>indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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				<p>Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.</p>	
20	HCEEE79	137	<p>Regulation of apoptosis of immune cells (such as mast cells).</p>	<p>Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate caspase protease-mediated apoptosis in immune cells (such as, for example, in mast cells). Mast cells are found in connective and mucosal tissues throughout the body, and their activation via immunoglobulin E -antigen, promoted by T helper cell type 2 cytokines, is an important component of allergic disease.</p>	<p>Preferred embodiments of the invention include using polypeptides of the invention (or antibodies, agonists, or antagonists thereof) in detection, diagnosis, prevention, and/or treatment of asthma, allergy, hypersensitivity and inflammation.</p>

					<p>Dysregulation of mast cell apoptosis may play a role in allergic disease and mast cell tumor survival. Exemplary assays for caspase apoptosis that may be used or routinely modified to test caspase apoptosis activity induced by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Masuda A, et al., <i>J Biol Chem</i>, 276(28):26107-26113 (2001); Yeatman CF 2nd, et al., <i>J Exp Med</i>, 192(8):1093-1103 (2000); Lee et al., <i>FEBS Lett</i> 485(2-3): 122-126 (2000); Nor et al., <i>J Vasc Res</i> 37(3): 209-218 (2000); and Karsan and Harlan, <i>J Atheroscler Thromb</i> 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Immune cells that may be used according to these assays are publicly available (e.g., through commercial sources). Exemplary immune cells that may be used according to these assays include mast cells such as the HMC human mast cell line.</p>	
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20	HCEEE79	137	Production of RANTES in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))	<p>RANTES FMAT. Assays for immunomodulatory proteins that induce chemotaxis of T cells, monocytes, and eosinophils are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, induce chemotaxis, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as RANTES, and the induction of chemotactic responses in immune cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Cocchi et al., Science</p>	
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				<p>270(5243):1811-1815 (1995); and Robinson et al., Clin Exp Immunol 101(3):398-407 (1995), the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are endothelial cells which line venous blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.</p>	
20	HCEEE79	137	Production of IL-10 and activation of T-cells.	<p>Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells.</p> <p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease,</p>	

				<p>Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary</p>	<p>multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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				T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
21	HCEFZ82	138	Production of IL-6	IL-6 F/MAT. IL-6 is produced by T cells and has strong effects on B cells. IL-6 participates in IL-4 induced IgE production and increases IgA production (IgA plays a role in mucosal immunity). IL-6 induces cytotoxic T cells. Deregulated expression of IL-6 has been linked to autoimmune disease, plasmacytomas, myelomas, and chronic hyperproliferative diseases. Assays for immunomodulatory and differentiation factor proteins produced by a large variety of cells where the expression level is strongly regulated by cytokines, growth factors, and hormones are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation and	A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-6 production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-6 production. A highly preferred indication is the stimulation or enhancement of mucosal immunity. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., as described below under "Infectious Disease"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Highly

				<p>differentiation and modulate T cell proliferation and function. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-6, and the stimulation and upregulation of T cell proliferation and functional activities. Such assays that may be used or routinely modified to test immunomodulatory and differentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); and Verhasselt et al., J Immunol 158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are</p>	<p>preferred indications also include boosting a B cell-mediated immune response and alternatively suppressing a B cell-mediated immune response. Highly preferred indications include inflammation and inflammatory disorders. Additional highly preferred indications include asthma and allergy. Highly preferred indications include neoplastic diseases (e.g., myeloma, plasmacytoma, leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, myeloma, plasmacytoma, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia,</p>
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				antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.	Hodgkin's disease, acute lymphocytic anemia (ALL), multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").
21	HCEFZ82	138	Production of IL-10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to	Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below),

				<p>assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2</p>	<p>immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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22	HCUCF89	139	Protection from Endothelial Cell Apoptosis.	polarizing conditions using peripheral blood lymphocytes isolated from cord blood. Caspase Apoptosis Rescue. Assays for caspase apoptosis rescue are well known in the art and may be used or routinely modified to assess the ability of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to inhibit caspase protease-mediated apoptosis. Exemplary assays for caspase apoptosis that may be used or routinely modified to test caspase apoptosis rescue of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Romeo et al., Cardiovasc Res 45(3): 788-794 (2000); Messmer et al., Br J Pharmacol 127(7): 1633-1640 (1999); and J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays are publicly	A highly preferred embodiment of the invention includes a method for stimulating endothelial cell growth. An alternative highly preferred embodiment of the invention includes a method for inhibiting endothelial cell growth. A highly preferred embodiment of the invention includes a method for stimulating endothelial cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting endothelial cell proliferation. A highly preferred embodiment of the invention includes a method for stimulating endothelial cell growth. An alternative highly preferred embodiment of the invention includes a method for inhibiting endothelial cell growth. A highly preferred embodiment of the invention includes a method for stimulating apoptosis of endothelial cells. An alternative
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				<p>available (e.g., through commercial sources). Exemplary endothelial cells that may be used according to these assays include bovine aortic endothelial cells (bAEC), which are an example of endothelial cells which line blood vessels and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.</p>	<p>highly preferred embodiment of the invention includes a method for inhibiting (e.g., decreasing) apoptosis of endothelial cells. A highly preferred embodiment of the invention includes a method for stimulating angiogenesis. An alternative highly preferred embodiment of the invention includes a method for inhibiting angiogenesis. A highly preferred embodiment of the invention includes a method for reducing cardiac hypertrophy. An alternative highly preferred embodiment of the invention includes a method for inducing cardiac hypertrophy. Highly preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), and disorders of the cardiovascular system (e.g., heart disease, congestive heart failure, hypertension, aortic stenosis, cardiomyopathy, valvular regurgitation, left ventricular dysfunction, atherosclerosis and atherosclerotic vascular disease, diabetic nephropathy,</p>
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				<p>intracardiac shunt, cardiac hypertrophy, myocardial infarction, chronic hemodynamic overload, and/or as described below under “Cardiovascular Disorders”). Highly preferred indications include cardiovascular, endothelial and/or angiogenic disorders (e.g., systemic disorders that affect vessels such as diabetes mellitus, as well as diseases of the vessels themselves, such as of the arteries, capillaries, veins and/or lymphatics). Highly preferred are indications that stimulate angiogenesis and/or cardiovascularization. Highly preferred are indications that inhibit angiogenesis and/or cardiovascularization.</p> <p>Highly preferred indications include antiangiogenic activity to treat solid tumors, leukemias, and Kaposi's sarcoma, and retinal disorders. Highly preferred indications include neoplasms and cancer, such as, Kaposi's sarcoma, hemangioma (capillary and cavernous), glomus tumors, telangiectasia, bacillary angiomatosis,</p>
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				<p>hemangioendothelioma, angiosarcoma, haemangiopericytoma, lymphangioma, lymphangiosarcoma. Highly preferred indications also include cancers such as, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Highly preferred indications also include arterial disease, such as, atherosclerosis, hypertension, coronary artery disease, inflammatory vasculitides, Reynaud's disease and Reynaud's phenomenon, aneurysms, restenosis; venous and lymphatic disorders such as thrombophlebitis, lymphangitis, and lymphedema; and other vascular disorders such as peripheral vascular disease, and cancer. Highly preferred indications also include trauma such as wounds, burns, and injured tissue (e.g., vascular injury such as, injury resulting</p>
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					<p>from balloon angioplasty, and atherosclerotic lesions), implant fixation, scarring, ischemia reperfusion injury, rheumatoid arthritis, cerebrovascular disease, renal diseases such as acute renal failure, and osteoporosis. Additional highly preferred indications include stroke, graft rejection, diabetic or other retinopathies, thrombotic and coagulative disorders, vasculitis, lymph angiogenesis, sexual disorders, age-related macular degeneration, and treatment /prevention of endometriosis and related conditions. Additional highly preferred indications include fibromas, heart disease, cardiac arrest, heart valve disease, and vascular disease. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple</p>
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22	HCUCF89	139	Regulation of apoptosis of immune cells (such as mast cells).	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate caspase protease-mediated apoptosis in immune cells (such as, for example, in mast cells). Mast cells are found in connective and mucosal tissues throughout the body, and their activation via immunoglobulin E -antigen, promoted by T helper cell type 2 cytokines, is an important component of allergic disease. Dysregulation of mast cell apoptosis may play a role in allergic disease and mast cell	<p>sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional preferred indications include inflammation and inflammatory disorders (such as acute and chronic inflammatory diseases, e.g., inflammatory bowel disease and Crohn's disease), and pain management.</p> <p>Preferred embodiments of the invention include using polypeptides of the invention (or antibodies, agonists, or antagonists thereof) in detection, diagnosis, prevention, and/or treatment of asthma, allergy, hypersensitivity and inflammation.</p>
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				tumor survival. Exemplary assays for caspase apoptosis that may be used or routinely modified to test caspase apoptosis activity induced by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Masuda A, et al., J Biol Chem, 276(28):26107-26113 (2001); Yeatman CF 2nd, et al., J Exp Med, 192(8):1093-1103 (2000); Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Immune cells that may be used according to these assays are publicly available (e.g., through commercial sources). Exemplary immune cells that may be used according to these assays include mast cells such as the HMC human mast cell line.	
	HCUCF89	139	Proliferation of pre-adipose cells (such	Assays for the regulation (i.e. increases or decreases) of	

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			as 3T3-L1 cells)	<p>viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of pre-adipose cells and cell lines. For example, the CellTiter-Glo[®] Luminescent Cell Viability Assay (Promega Corp., Madison, WI, USA) can be used to measure the number of viable cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells. 3T3-L1 is a mouse preadipocyte cell line. It is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation. Cells were differentiated to an adipose-like state before being used in the screen. See Green H and Meuth M., Cell 3: 127-133 (1974), which is herein incorporated by reference in its entirety.</p>	
23	HCWAE64	140	Regulation of transcription via	Assays for the regulation of transcription through the	A highly preferred indication is diabetes mellitus. Additional

			<p>DMEF1 response element in adipocytes and pre-adipocytes</p>	<p>DMEF1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the DMEF1 response element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insulin production. The DMEF1 response element is present in the GLUT4 promoter and binds to MEF2 transcription factor and another transcription factor that is required for insulin regulation of Glut4 expression in skeletal muscle. GLUT4 is the primary insulin-responsive glucose transporter in fat and muscle tissue. Exemplary assays that may be used or routinely modified to test for DMEF1 response element activity (in adipocytes and pre-adipocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Thai, M.V., et al., J Biol Chem, 273(23):14285-92</p>	<p>highly preferred indications include complications associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness),</p>
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				<p>(1998); Mora, S., et al., J Biol Chem, 275(21):16323-8 (2000); Liu, M.L., et al., J Biol Chem, 269(45):28514-21 (1994); "Identification of a 30-base pair regulatory element and novel DNA binding protein that regulates the human GLUT4 promoter in transgenic mice", J Biol Chem. 2000 Aug 4;275(31):23666-73; Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Adipocytes and pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include the mouse 3T3-L1 cell line which is an adherent mouse preadipocyte cell line. Mouse 3T3-L1 cells are a continuous substrain of 3T3 fibroblasts developed through clonal isolation. These cells undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation</p>	<p>ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance.</p>
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23	HCWAE64	140	<p>Activation of transcription through cAMP response element (CRE) in pre-adipocytes.</p>	<p>culture conditions.</p> <p>Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies</p>	<p>A highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. An additional highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyposmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease,</p>
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			<p>and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.</p>	<p>hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). Additional highly preferred indications are complications associated with insulin resistance.</p>
HCWAE64	140	Activation of	Assays for the activation of	A highly preferred indication is

23	transcription through serum response element in pre-adipocytes.	transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through	obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. An additional highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hypermolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular
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				<p>the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.</p>	<p>Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below). Additional highly preferred indications are complications associated with insulin resistance.</p>
23	HCWAE64	140	<p>Activation of transcription through GATA-3 response element in immune cells (such as mast cells).</p>	<p>This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the</p>	<p>Highly preferred indications include allergy, asthma, and rhinitis. Additional preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications also include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g.,</p>

				<p>invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these</p>	<p>rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary tract cancers and/or as described below under "Hyperproliferative Disorders"). Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia,</p>
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				assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.
23	HCWAE64	140	Activation of transcription through NFAT response element in immune cells (such as mast cells).	<p>This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes</p>	<p>Highly preferred indications include allergy, asthma, and rhinitis. Additional preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications also include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies</p>

				involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1	(e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary tract cancers and/or as described below under "Hyperproliferative Disorders"). Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.
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				cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	
23	HCWAE64	140	Activation of transcription through NFAT response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and	Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").

			<p>Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.</p>	<p>Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes</p>
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23	HCWAE64	140	Activation of transcription through serum response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et	meilitus, endocarditis, meningitis, Lyme Disease, asthma and allergy. A preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) TNF alpha production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) TNF alpha production. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and
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				<p>al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.</p>	<p>inflammatory disorders, and treating joint damage in patients with rheumatoid arthritis. An additional highly preferred indication is sepsis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Additionally, highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, glioma (e.g., malignant glioma), solid tumors, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS,</p>
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					<p>granulomatous disease, inflammatory bowel disease, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, cardiac reperfusion injury, and asthma and allergy. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").</p>
23	HCWAE64	140	<p>Activation of transcription through GAS response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that</p>	<p>Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma (e.g., T cell lymphoma, Burkitt's lymphoma, non-Hodgkins lymphoma, Hodgkin's disease), melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include</p>

			<p>may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Hentinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the SUPT cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).</p>	<p>benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., viral infections, tuberculosis, infections associated with chronic granulomatous disease and malignant osteoporosis, and/or an infectious disease as described below under "Infectious Disease"). An additional preferred indication is</p>
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					<p>idiopathic pulmonary fibrosis. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, and asthma and allergy.</p>
24	HCWUL09	141	<p>Activation of transcription through GATA-3 response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of genes important for Th2 immune response development.</p>	<p>A highly preferred indication includes allergy. A highly preferred indication includes asthma. A highly preferred indication includes rhinitis. Additional highly preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described</p>

				<p>Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the HT2 cell line,</p>	<p>below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancer, such as, for example, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias,</p>
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				<p>which is a suspension culture of IL-2 dependent T cells that also respond to IL-4.</p>	<p>Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
25	HDPDI72	142	<p>Activation of transcription through serum response element in immune cells (such as natural killer cells).</p>	<p>Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE</p>	<p>A preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) TNF alpha production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) TNF alpha production. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis,</p>

				<p>activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.</p>	<p>systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders, and treating joint damage in patients with rheumatoid arthritis. An additional highly preferred indication is sepsis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Additionally, highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, glioma (e.g., malignant glioma), solid tumors, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative</p>
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					disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, cardiac reperfusion injury, and asthma and allergy. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").
25	HDPDI72	142	Production of IL-10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention	Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune

				<p>(including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6.</p>	<p>Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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26	HDPFY18	143		<p>Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.</p> <p>TNFα FMT. Assays for immunomodulatory proteins produced by activated macrophages, T cells, fibroblasts, smooth muscle, and other cell types that exert a wide variety of inflammatory and cytotoxic effects on a variety of cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, modulate inflammation and cytotoxicity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines such as tumor necrosis factor alpha (TNFα),</p>	<p>A highly preferred embodiment of the invention includes a method for inhibiting (e.g., decreasing) TNFα production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) TNFα production. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies</p>
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			and the induction or inhibition of an inflammatory or cytotoxic response. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Verhasselt et al., Eur J Immunol 28(11):3886-3890 (1998); Dahlen et al., J Immunol 160(7):3585-3593 (1998); Verhasselt et al., J Immunol 158:2919-2925 (1997); and Nardelli et al., J Leukoc Biol 65:822-828 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or	(e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders, and treating joint damage in patients with rheumatoid arthritis. An additional highly preferred indication is sepsis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Additionally, highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, melanoma, glioma (e.g., malignant glioma), solid tumors, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia,
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				cytokines, initiate and upregulate T cell proliferation and functional activities.	pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, cardiac reperfusion injury, and asthma and allergy. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").
26	HDPFY18	143	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and	Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases

			<p>modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFkB response element that may be used or routinely modified to test NFkB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>(e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's</p>
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					<p>disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.</p>
27	HDPIE44	144	<p>Activation of JNK Signaling Pathway in immune cells (such as eosinophils).</p>	<p>Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and</p>	<p>Highly preferred indications include asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below). Highly preferred indications also</p>

				<p>agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils.</p> <p>Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include</p>	<p>include boosting or inhibiting immune cell proliferation. Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.</p>
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				assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.	
				Assays for the regulation of transcription of Malic Enzyme are well-known in the art and may be used or routinely modified to assess the ability of	
				Regulation of transcription of Malic Enzyme in hepatocytes	
				145	
				HDPIU94	
28					A highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g.,

			<p>polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate transcription of Malic Enzyme, a key enzyme in lipogenesis. Malic enzyme is involved in lipogenesis and its expression is stimulated by insulin. ME promoter contains two direct repeat (DR1)-like elements MEp and MEed identified as putative PPAR response elements. ME promoter may also respond to AP1 and other transcription factors. Exemplary assays that may be used or routinely modified to test for regulation of transcription of Malic Enzyme (in hepatocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Streeper, R.S., et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol, 8(10):1361-9 (1994); Barroso, I., et al., J Biol Chem, 274(25):17997-8004 (1999); Ijpenberg, A., et al., J Biol Chem, 272(32):20108-</p>	<p>diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyposmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders</p>
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				<p>20117 (1997); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays includes the mouse 3T3-L1 cell line. 3T3-L1 is a mouse preadipocyte cell line (adherent). It is a continuous substrain of 3T3 fibroblasts developed through clonal isolation. Cells undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation culture conditions.</p>	<p>as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance.</p>
28	HDPIU94	145	<p>Activation of transcription through NFKB response element in immune cells (such as EOL1 cells).</p>	<p>Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and</p>	<p>Highly preferred indications include asthma, allergy, hypersensitivity reactions, and inflammation. Preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"),</p>

				<p>agonists or antagonists of the invention) to regulate NFkB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFkB response element that may be used or routinely modified to test NFkB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Valle Blazquez et al, Immunology 90(3):455-460 (1997); Aramburau et al., J Exp Med 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. For example, a reporter assay (which measures increases in transcription inducible from a NFkB responsive element in EOL-1 cells) may link the</p>	<p>immunological disorders, inflammation and inflammatory disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below).</p>
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				NFKB element to a reporter gene and binds to the NFKB transcription factor, which is upregulated by cytokines and other factors. Exemplary immune cells that may be used according to these assays include eosinophils such as the human EOL-1 cell line of eosinophils. Eosinophils are a type of immune cell important in the allergic responses; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Eol-1 is a human eosinophil cell line.	
				Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal transduction that regulate cell proliferation or differentiation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity	
	HDPIU94	145	Activation of Hepatocyte ERK Signaling Pathway		A highly preferred embodiment of the invention includes a method for stimulating hepatocyte cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting hepatocyte cell proliferation. A highly preferred embodiment of the invention includes a method for stimulating hepatocyte cell differentiation. An alternative highly preferred embodiment of the invention includes a method

				<p>that may be used or routinely modified to test ERK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Rat liver hepatoma cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary rat liver hepatoma cells that may be used according to these assays include H4Ile cells, which are known to respond to glucocorticoids, insulin, or cAMP derivatives.</p>	<p>for inhibiting hepatocyte cell differentiation. A highly preferred embodiment of the invention includes a method for activating hepatocyte cells. An alternative highly preferred embodiment of the invention includes a method for inhibiting the activation of and/or inactivating hepatocyte cells. Highly preferred indications include disorders of the liver and/or endocrine disorders (e.g., as described below under "Endocrine Disorders"). Preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), immune disorders (e.g., as described below under "Immune Activity"), neural disorders (e.g., as described below under "Neural Activity and Neurological Diseases"), and infection (e.g., as described below under "Infectious Disease"). A highly</p>
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					<p>preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision</p>
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					<p>impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance. Additional highly preferred indications are disorders of the musculoskeletal systems including myopathies, muscular dystrophy, and/or as described herein. Additional highly preferred indications include, hepatitis, jaundice, gallstones, cirrhosis of the liver, degenerative or necrotic liver disease, alcoholic liver diseases, fibrosis, liver regeneration, metabolic disease,</p>
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					<p>dyslipidemia and cholesterol metabolism.</p> <p>Additional highly preferred indications include neoplasms and cancers, such as, hepatocarcinomas, other liver cancers, and colon and pancreatic cancer. Preferred indications also include prostate, breast, lung, esophageal, stomach, brain, and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.</p>
28	HDPIU94	145	<p>Regulation of proliferation and/or differentiation in immune cells (such as mast cells).</p>	<p>Kinase assays, for example an Elk-1 kinase assay for ERK signal transduction that regulates cell proliferation or differentiation, are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used</p>	<p>Preferred embodiments of the invention include using polypeptides of the invention (or antibodies, agonists, or antagonists thereof) in detection, diagnosis, prevention, and/or treatment of asthma, allergy, hypersensitivity and inflammation.</p>

				or routinely modified to test ERK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Ali H, et al., J Immunol, 165(12):7215-7223 (2000); Tam SY, et al., Blood, 90(5):1807-1820 (1997); Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Berra et al., Biochem Pharmacol 60(8):1171-1178 (2000); Gupta et al., Exp Cell Res 247(2):495-504 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary immune cells that may be used according to these assays include human mast cells such as the HMC-1 cell line.	
				IFNgamma F ₁ MAT. IFN γ plays a central role in the immune system and is considered to be a proinflammatory cytokine. IFN γ promotes TH1 and inhibits TH2 differentiation;	A highly preferred embodiment of the invention includes a method for stimulating the production of IFN γ . An alternative highly preferred embodiment of the
28	HDPIU94	145	Production of IFNgamma using a T cells		

				<p>promotes IgG2a and inhibits IgE secretion; induces macrophage activation; and increases MHC expression. Assays for immunomodulatory proteins produced by T cells and NK cells that regulate a variety of inflammatory activities and inhibit TH2 helper cell functions are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, regulate inflammatory activities, modulate TH2 helper cell function, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as Interferon gamma (IFNg), and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the</p>	<p>invention includes a method for inhibiting the production of IFNg. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., viral infections, tuberculosis, infections associated with chronic granulomatous disease and malignant osteoporosis, and/or as described below under "Infectious Disease"). Highly preferred indications include autoimmune disease (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiency (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. Additional preferred indications include idiopathic pulmonary fibrosis. Highly preferred indications include</p>
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				<p>invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Gonzalez et al., J Clin Lab Anal 8(5):225-233 (1995); Billiau et al., Ann NY Acad Sci 856:22-32 (1998); Boehm et al., Annu Rev Immunol 15:749-795 (1997), and Rheumatology (Oxford) 38(3):214-20 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T Cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.</p>	<p>neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes</p>
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					mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.
29	HDPPD93	146	Inhibition of adipocyte ERK signaling pathway.	<p>Kinase assay: measures the phosphorylation of Elk-1, an indication of activation of extracellular signal regulated kinase (ERK). ERK pathway regulates cell growth, proliferation and differentiation. Cells were pretreated with SID supernatants for 15-18 hours, and then 100 nM of insulin was added to stimulate ERK kinase. Phosphorylation of Elk-1 was measured after a 20 minute incubation. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated.</p> <p>Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation</p>	

29	HDPPD93	146	Activation of Adipocyte PI3 Kinase Signalling Pathway	<p>conditions known in the art. Cells were differentiated to an adipose-like state before being used in the screen. See Green et al., Cell 3: 127-133 (1974), the contents of which are herein incorporated by reference in its entirety.</p> <p>Kinase assay. Kinase assays, for example an GSK-3 assays, for PI3 kinase signal transduction that regulate glucose metabolism and cell survival are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit glucose metabolism and cell survival. Exemplary assays for PI3 kinase activity that may be used or routinely modified to test PI3 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Nikoulina et al.,</p>	<p>A highly preferred embodiment of the invention includes a method for increasing adipocyte survival. An alternative highly preferred embodiment of the invention includes a method for decreasing adipocyte survival. A preferred embodiment of the invention includes a method for stimulating adipocyte proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting adipocyte proliferation. A preferred embodiment of the invention includes a method for stimulating adipocyte differentiation. An alternative highly preferred embodiment of the invention includes a method for inhibiting adipocyte differentiation. Highly</p>
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				<p>Diabetes 49(2):263-271 (2000); and Schreyer et al., Diabetes 48(8):1662-1666 (1999), the contents of each of which are herein incorporated by reference in its entirety. Mouse adipocyte cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.</p>	<p>preferred indications include endocrine disorders (e.g., as described below under "Endocrine Disorders"). Preferred indications include neoplastic diseases (e.g., lipomas, liposarcomas, and/or as described below under "Hyperproliferative Disorders"), blood disorders (e.g., hypertension, congestive heart failure, blood vessel blockage, heart disease, stroke, impotence and/or as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), immune disorders (e.g., as described below under "Immune Activity"), neural disorders (e.g., as described below under "Neural Activity and Neurological Diseases"), and infection (e.g., as described below under "Infectious Disease"). A highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease</p>
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					<p>(e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below,</p>
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				<p>especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance. Additional highly preferred indications are disorders of the musculoskeletal systems including myopathies, muscular dystrophy, and/or as described herein.</p> <p>Additional highly preferred indications include, hypertension, coronary artery disease, dyslipidemia, gallstones, osteoarthritis, degenerative arthritis, eating disorders, fibrosis, cachexia, and kidney diseases or disorders. Highly preferred indications include neoplasms and cancer, such as, lipoma, liposarcoma, lymphoma, leukemia and breast, colon, and kidney cancer. Additional highly preferred</p>
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29	HDPPD93	146	Activation of transcription through API response element in immune cells (such as T-cells).	Assays for the activation of transcription through the API response element are known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the API response element that may be used or routinely modified to test API-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol	indications include melanoma, prostate, lung, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.
				Preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), and infection (e.g., an infectious disease as described below under "Infectious Disease"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional highly preferred indications include inflammation and	

				<p>216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Mouse T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the HT2 cell line, which is an IL-2 dependent suspension culture cell line that also responds to IL-4.</p>	<p>inflammatory disorders. Highly preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include arthritis, asthma, AIDS, allergy, anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, granulomatous disease, inflammatory bowel disease, sepsis, psoriasis, suppression of immune reactions to transplanted organs and tissues, endocarditis, meningitis, and Lyme Disease.</p>
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29	HDPPD93	146	<p>Activation of transcription through NFAT response element in immune cells (such as natural killer cells).</p>	<p>Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999);</p>	<p>Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia,</p>
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				<p>Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.</p>	<p>lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.</p>
29	HDPPD93	146	<p>Activation of transcription through API response element in immune</p>	<p>Assays for the activation of transcription through the API response element are well-known in the art and may be</p>	<p>Preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"),</p>

			cells (such as T-cells).	<p>used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the API response element that may be used or routinely modified to test API-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays are publicly</p>	<p>blood disorders (e.g., as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), and infection (e.g., an infectious disease as described below under "Infectious Disease"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic</p>
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				<p>available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is an IL-2 and IL-4 responsive suspension-culture cell line.</p>	<p>conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include arthritis, asthma, AIDS, allergy, anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, granulomatous disease, inflammatory bowel disease, sepsis, psoriasis, suppression of immune reactions to transplanted organs and tissues, endocarditis, meningitis, and Lyme Disease.</p>
29	HDPD93	146	<p>Activation of transcription through CD28 response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the CD28 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T cells. Exemplary assays for transcription through the CD28 response element that may be used or routinely modified to</p>	<p>A highly preferred embodiment of the invention includes a method for stimulating T cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting T cell proliferation. A highly preferred embodiment of the invention includes a method for activating T cells. An alternative highly preferred embodiment of the invention includes a method for inhibiting the activation of and/or inactivating T cells.</p>

				<p>test CD28-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol 159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-2 production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-2 production. Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Highly preferred indications include neoplastic diseases (e.g., melanoma, renal cell carcinoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, melanoma (e.g., metastatic melanoma), renal cell carcinoma (e.g.,</p>
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					<p>metastatic renal cell carcinoma), leukemia, lymphoma (e.g., T cell lymphoma), and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. A highly preferred indication includes infection (e.g., AIDS, tuberculosis, infections associated with granulomatous disease, and osteoporosis, and/or as described below under "Infectious Disease"). A highly preferred indication is AIDS. Additional highly preferred indications include suppression of immune reactions to transplanted organs and/or tissues, uveitis, psoriasis, and tropical spastic paraparesis. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications also</p>
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29	HDPPD93	146	<p>Activation of transcription through NFAT response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to</p>	<p>include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.</p>	<p>Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred</p>
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				<p>test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>indications include inflammation and inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease,</p>
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29	HDPPD93	146	<p>Activation of transcription through NFKB response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays</p>	<p>inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.</p> <p>Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred</p>
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				<p>disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease,</p>
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29	HDPDP93	146	Production of IL-10 and activation of T-cells.	<p>Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells.</p> <p>Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88:187-196 (2000); the contents</p>	<p>suppression of immune reactions to transplanted organs, asthma and allergy.</p> <p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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				of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
30	HDTLM18	147	Production of MIP1alpha	MIP-1alpha FMAT. Assays for immunomodulatory proteins produced by activated dendritic cells that upregulate monocyte/macrophage and T cell chemotaxis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate	A highly preferred embodiment of the invention includes a method for stimulating MIP1a production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) MIP1a production. A highly preferred indication is infection (e.g., an infectious disease as described below under

				<p>immunomodulation, modulate chemotaxis, and modulate T cell differentiation. Exemplary assays that test for immunomodulatory proteins evaluate the production of chemokines, such as macrophage inflammatory protein 1 alpha (MIP-1a), and the activation of monocytes/macrophages and T cells. Such assays that may be used or routinely modified to test immunomodulatory and chemotaxis activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Sathaporn and Eremin, J R Coll Surg Ednb 45(1):9-19 (2001); Drakes et al., Transp Immunol 8(1):17-29 (2000); Verhasselt et al., J Immunol 158:2919-2925 (1997); and Nardelli et al., J Leukoc Biol 65:822-828 (1999), the contents of each of which are herein</p>	<p>"Infectious Disease"). Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional highly preferred indications include inflammation and inflammatory disorders. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes</p>
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				<p>incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.</p>	<p>mellitus, endocarditis, meningitis, Lyme Disease, asthma, and allergy. Preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.</p>
30	HDTLM18	147	<p>Production of IL-13 and activation of T-cells.</p>	<p>Assays for production of IL-13 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-13 and/or activation of T-cells. Exemplary assays for IL-13</p>	<p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as</p>

				<p>production that may be used or routinely modified to test activity of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) include, for example, assays such as disclosed and/or cited in: Grunig, G, et al., "Requirement for IL-13 independently of IL-4 in Experimental asthma" Science; 282: 2261-2263 (1998), and Wills-Karp M, et al., "Interleukin-13: central mediator of allergic asthma" Science; 282: 2258-2261 (1998); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL13, a Th2 type cytokine, is a potent stimulus for mucus production, airway hyper-responsiveness and allergic asthma. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis</p>	<p>described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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				of allergy and asthma. Primary T helper 2 cells are generated in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
31	HE6CS65	148	Production of IL-6	IL-6 FMT. IL-6 is produced by T cells and has strong effects on B cells. IL-6 participates in IL-4 induced IgE production and increases IgA production (IgA plays a role in mucosal immunity). IL-6 induces cytotoxic T cells. Deregulated expression of IL-6 has been linked to autoimmune disease, plasmacytomas, myelomas, and chronic hyperproliferative diseases. Assays for immunomodulatory and differentiation factor proteins produced by a large variety of cells where the expression level is strongly regulated by cytokines, growth factors, and hormones are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate	A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-6 production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-6 production. A highly preferred indication is the stimulation or enhancement of mucosal immunity. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., as described below under "Infectious Disease"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as

			<p>immunomodulation and differentiation and modulate T cell proliferation and function. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-6, and the stimulation and upregulation of T cell proliferation and functional activities. Such assays that may be used or routinely modified to test immunomodulatory and differentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); and Verhasselt et al., J Immunol 158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art.</p>	<p>described below). Highly preferred indications also include boosting a B cell-mediated immune response and alternatively suppressing a B cell-mediated immune response. Highly preferred indications include inflammation and inflammatory disorders. Additional highly preferred indications include asthma and allergy. Highly preferred indications include neoplastic diseases (e.g., myeloma, plasmacytoma, leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, myeloma, plasmacytoma, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia,</p>
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				Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.	leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").
31	HE6CS65	148	Production of MCP-1	MCP-1 FMAT. Assays for immunomodulatory proteins that are produced by a large variety of cells and act to induce chemotaxis and activation of monocytes and T cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the	A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) MCP-1 production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) MCP-1 production. A highly preferred indication is infection (e.g., an infectious disease as

				<p>invention) to mediate immunomodulation, induce chemotaxis, and modulate immune cell activation. Exemplary assays that test for immunomodulatory proteins evaluate the production of cell surface markers, such as monocyte chemoattractant protein (MCP), and the activation of monocytes and T cells. Such assays that may be used or routinely modified to test immunomodulatory and differentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Sathaporn and Eremin, J R Coll Surg Ednb 45(1):9-19 (2001); and Verhasselt et al., J Immunol 158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these</p>	<p>described below under "Infectious Disease"). Additional highly preferred indications include inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs</p>
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				assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.	and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis (bacterial and viral), Lyme Disease, asthma, and allergy Preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.
31	HE6CS65	148	Production of IL-10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or	Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus

				<p>activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis</p>	<p>erythematosis, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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32	HE8BQ49	149	Inhibition of adipocyte ERK signaling pathway.	<p>of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.</p> <p>Kinase assay: measures the phosphorylation of Elk-1, an indication of activation of extracellular signal regulated kinase (ERK). ERK pathway regulates cell growth, proliferation and differentiation. Cells were pretreated with SID supernatants for 15-18 hours, and then 100 nM of insulin was added to stimulate ERK kinase. Phosphorylation of Elk-1 was measured after a 20 minute incubation. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and</p>	
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32	HE8BQ49	149	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art. Cells were differentiated to an adipose-like state before being used in the screen. See Green et al., Cell 3: 127-133 (1974), the contents of which are herein incorporated by reference in its entirety.	Highly preferred indications include asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below). Highly preferred indications also include boosting or inhibiting immune cell proliferation. Preferred indications include
				Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol	

				<p>Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-</p>	<p>neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.</p>
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				induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.	
32	HE8BQ49	149	Production of RANTES in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))	RANTES FMAT. Assays for immunomodulatory proteins that induce chemotaxis of T cells, monocytes, and eosinophils are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the	

				<p>invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, induce chemotaxis, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as RANTES, and the induction of chemotactic responses in immune cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Cocchi et al., Science 270(5243):1811-1815 (1995); and Robinson et al., Clin Exp Immunol 101(3):398-407 (1995), the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays</p>	
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32	HE8BQ49	149	Production of VCAM in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))	are publicly available (e.g., through the ATCC). Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are endothelial cells which line venous blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Assays for measuring expression of VCAM are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate VCAM expression. For example, FMAT may be used to measure the upregulation of cell surface VCAM-1 expression in endothelial cells. Endothelial cells are cells that line blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular	Highly preferred indications include inflammation (acute and chronic), restenosis, atherosclerosis, asthma and allergy. Highly preferred indications include inflammation and inflammatory disorders, immunological disorders, neoplastic disorders (e.g. cancer/tumorigenesis), and cardiovascular disorders (such as described below under "Immune Activity", "Blood-Related Disorders", "Hyperproliferative Disorders" and/or "Cardiovascular Disorders"). Highly preferred indications include neoplasms and cancers such as, for
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				tone, and immune cell extravasation. Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are available from commercial sources. The expression of VCAM (CD106), a membrane-associated protein, can be upregulated by cytokines or other factors, and contributes to the extravasation of lymphocytes, leucocytes and other immune cells from blood vessels; thus VCAM expression plays a role in promoting immune and inflammatory responses.	example, leukemia, lymphoma, melanoma, renal cell carcinoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.
32	HE8BQ49	149	Activation of Transcription	Assays for activation of transcription are well-known in the art and may be used and routinely modified to assess ability of polypeptides of the invention to inhibit or activate transcription. An example of such an assay follows: Cells were pretreated with SID supernatants or controls for 15-18 hours. SEAP activity was measured after 48 hours. LS174T is an epithelial colon	

33	HE9CY05	150	<p>adenocarcinoma cell line. Its tumorigenicity in nude mice make cell line LS174T a model for studies on the mechanism of synthesis and secretion of specific tumoral markers in colon cancer. See, Patan et al., Circ Res, 89(8):732-39 (2001), the contents of which are herein incorporated by reference in its entirety.</p> <p>Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of genes important for Th2 immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention</p>	<p>A highly preferred indication includes allergy. A highly preferred indication includes asthma. A highly preferred indication includes rhinitis. Additional highly preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic</p>
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				<p>(including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the HT2 cell line, which is a suspension culture of IL-2 dependent T cells that also respond to IL-4.</p>	<p>lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancer, such as, for example, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia,</p>
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34	HEAA W94	151	Production of IL-10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-	neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease. Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.
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				<p>helper type 2 cell-directed therapy for asthma"</p> <p>Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.</p>	
35	HEBFR46	152	<p>Activation of transcription through cAMP response element (CRE) in pre-adipocytes.</p>	<p>Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and</p>	<p>A highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. An additional highly preferred indication is diabetes mellitus.</p>

				<p>agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al.,</p>	<p>An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness),</p>
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				<p>J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.</p>	<p>ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). Additional highly preferred indications are complications associated with insulin resistance.</p>
35	HEBFR46	152	<p>Activation of transcription through GATA-3 response element in immune cells (such as mast cells).</p>	<p>This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may</p>	<p>Highly preferred indications include allergy, asthma, and rhinitis. Additional preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications also include blood disorders (e.g., as described below under</p>

				<p>be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol</p>	<p>"Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary tract cancers and/or as described below under "Hyperproliferative Disorders"). Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel</p>
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				<p>14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.</p>	<p>disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
35	HEBFR46	152	<p>Stimulation of insulin secretion from pancreatic beta cells.</p>	<p>Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and</p>	<p>A highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel</p>

				<p>disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced</p>	<p>blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred</p>
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				<p>rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.</p>	<p>indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance.</p>
35	HEBFR46	152	<p>Activation of transcription through AP1 response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the AP1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the AP1 response element that may be used or routinely modified to test AP1-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA</p>	<p>Preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), and infection (e.g., an infectious disease as described below under "Infectious Disease"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional highly preferred indications include inflammatory and inflammatory disorders. Highly preferred indications also include neoplastic diseases</p>

				<p>85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is an IL-2 and IL-4 responsive suspension-culture cell line.</p>	<p>(e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include arthritis, asthma, AIDS, allergy, anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, granulomatous disease, inflammatory bowel disease, sepsis, psoriasis, suppression of immune reactions to transplanted organs and tissues, endocarditis, meningitis, and Lyme Disease.</p>
35	HEBFR46	152	Activation of transcription through CD28 response	Assays for the activation of transcription through the CD28 response element are well-	<p>A highly preferred embodiment of the invention includes a method for</p>

			<p>known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T cells. Exemplary assays for transcription through the CD28 response element that may be used or routinely modified to test CD28-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol 159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly</p>	<p>stimulating T cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting T cell proliferation. A highly preferred embodiment of the invention includes a method for activating T cells. An alternative highly preferred embodiment of the invention includes a method for inhibiting the activation of and/or inactivating T cells. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-2 production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-2 production. Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-</p>
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				<p>available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>mediated immune response. Highly preferred indications include neoplastic diseases (e.g., melanoma, renal cell carcinoma, leukemia, lymphoma, and/or as described below under “Hyperproliferative Disorders”). Highly preferred indications include neoplasms and cancers, such as, for example, melanoma (e.g., metastatic melanoma), renal cell carcinoma (e.g., metastatic renal cell carcinoma), leukemia, lymphoma (e.g., T cell lymphoma), and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. A highly preferred indication includes infection (e.g., AIDS, tuberculosis, infections associated with granulomatous disease, and osteoporosis, and/or as described below under “Infectious Disease”). A highly preferred indication is AIDS. Additional highly preferred</p>
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					indications include suppression of immune reactions to transplanted organs and/or tissues, uveitis, psoriasis, and tropical spastic paraparesis. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.
35	HEBFR46	152	Activation of transcription through NFAT response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely	Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders").

				<p>modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by</p>	<p>Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative</p>
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				reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.	disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.
35	HEBFR46	152	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB	Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications

				<p>transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFkB response element that may be used or routinely modified to test NFkB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia,</p>
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					<p>thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.</p>
35	HEBFR46	152	<p>Activation of transcription through STAT6 response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely</p>	<p>A highly preferred indication is allergy. Another highly preferred indication is asthma. Additional highly preferred indications include inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple</p>

				<p>modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.</p> <p>Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune</p>
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37	HFCDW95	154	<p>Activation of transcription through cAMP response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, bind to CREB transcription factor, and modulate expression of genes involved in a wide variety of cell functions. Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of</p>	<p>reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").</p> <p>Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional preferred indications include inflammation and inflammatory</p>
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				<p>the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Genes 15(2):105-117 (1997); and Belkowski et al., J Immunol 161(2):659-665 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.</p>	<p>disorders. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma (e.g., T cell lymphoma, Burkitt's lymphoma, non-Hodgkins lymphoma, Hodgkin's disease), melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune</p>
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					reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, and asthma and allergy.
37	HFCDW95	154	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn	Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under

				<p>et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the MOLT4, that may be used according to these assays are publicly available (e.g., through the ATCC).</p>	<p>“Hyperproliferative Disorders”). Highly preferred indications include neoplasms and cancers, such as, for example, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin’s disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt’s lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.</p>
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38	HFEBO17	155	<p>Activation of JNK Signaling Pathway in immune cells (such as eosinophils).</p>	<p>Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety.</p>	<p>Highly preferred indications include asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below). Highly preferred indications also include boosting or inhibiting immune cell proliferation. Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.</p>
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				<p>Exemplary cells that may be used according to these assays include eosinophils.</p> <p>Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74;</p>	
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				and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.	
39	HFIJA29	156	Production of IL-4	IL-4 FMAT. Assays for immunomodulatory proteins secreted by TH2 cells that stimulate B cells, T cells, macrophages and mast cells and promote polarization of CD4+ cells into TH2 cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, stimulate immune cells, modulate immune cell polarization, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory	A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-4 production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-4 production. A highly preferred indication includes asthma. A highly preferred indication includes allergy. A highly preferred indication includes rhinitis. Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma,

			<p>proteins evaluate the production of cytokines, such as IL-4, and the stimulation of immune cells, such as B cells, T cells, macrophages and mast cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Gonzalez et al., J Clin Lab Anal 8(5):277-283 (1194); Yssel et al., Res Immunol 144(8):610-616 (1993); Bagley et al., Nat Immunol 1(3):257-261 (2000); and van der Graaff et al., Rheumatology (Oxford) 38(3):214-220 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are</p>	<p>melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute</p>
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				primary human lymphocytes that mature in the thymus and express a T cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.	lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").
40	HFKFX64	157	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be	Highly preferred indications include asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis

				<p>used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils.</p> <p>Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of</p>	<p>and/or as described below), immunodeficiencies (e.g., as described below). Highly preferred indications also include boosting or inhibiting immune cell proliferation. Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.</p>
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41	transcription through API response element in immune cells (such as T-cells).	transcription through the API response element are known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the API response element that may be used or routinely modified to test API-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Mouse T cells	neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), and infection (e.g., an infectious disease as described below under "Infectious Disease"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer.
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				that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the HT2 cell line, which is an IL-2 dependent suspension culture cell line that also responds to IL-4.	Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include arthritis, asthma, AIDS, allergy, anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, granulomatous disease, inflammatory bowel disease, sepsis, psoriasis, suppression of immune reactions to transplanted organs and tissues, endocarditis, meningitis, and Lyme Disease.
41	HGBER72	158	Activation of transcription through NFAT response in immune cells (such as T-cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and	Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below),

			<p>modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according</p>	<p>immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia,</p>
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				to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.	thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.
41	HGBER72	158	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be	Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as

				<p>used or routinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the MOLT4, that may be used according to these assays are publicly available (e.g., through the ATCC).</p>	<p>described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease,</p>
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					inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.
42	HGBGN34	159	Production of IL-5	IL-5 FMAT. Assays for immunomodulatory proteins secreted by TH2 cells, mast cells, basophils, and eosinophils that stimulate eosinophil function and B cell Ig production and promote polarization of CD4+ cells into TH2 cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, stimulate immune cell function, modulate B cell Ig production, modulate immune cell polarization, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins	<p>A highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-5 production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-5 production. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) immunoglobulin production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., decreasing) immunoglobulin production. A highly preferred indication includes allergy. A highly preferred indication includes asthma. A highly preferred indication includes rhinitis.</p>

			<p>evaluate the production of cytokines, such as IL-5, and the stimulation of eosinophil function and B cell Ig production. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Ohshima et al., Blood 92(9):3338-3345 (1998); Jung et al., Eur J Immunol 25(8):2413-2416 (1995); Mori et al., J Allergy Clin Immunol 106(1 Pt 2):558-564 (2000); and Koning et al., Cytokine 9(6):427-436 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human</p>	<p>An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative</p>
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				<p>lymphocytes that mature in the thymus and express a T cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.</p>	<p>disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
42	HGBGN34	159	Activation of Transcription	<p>Assays for activation of transcription are well-known in the art and may be used and routinely modified to assess ability of polypeptides of the invention to inhibit or activate transcription. An example of such an assay follows: Cells were pretreated with SID supernatants or controls for 15-18 hours. SEAP activity was measured after 48 hours.</p>	

43	HGLBG15	160			<p>LS174T is an epithelial colon adenocarcinoma cell line. Its tumorigenicity in nude mice make cell line LS174T a model for studies on the mechanism of synthesis and secretion of specific tumoral markers in colon cancer. See, Patan et al., Circ Res, 89(8):732-39 (2001), the contents of which are herein incorporated by reference in its entirety.</p> <p>Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays</p>	<p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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				such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
44	HHFEC39	161	Production of IL-10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may	Highly preferred indications include allergy and asthma. Additional highly preferred

				<p>be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of</p>	<p>indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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45	HHSD153	162	<p>Activation of transcription through serum response element in immune cells (such as T-cells).</p>	<p>Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.</p>	<p>Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or</p>	<p>A preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) TNF alpha production. An alternative preferred embodiment of the invention includes a method for stimulating (e.g., increasing) TNF alpha production. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus,</p>
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				<p>antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension culture of T cells with cytotoxic activity.</p>	<p>Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders, and treating joint damage in patients with rheumatoid arthritis. An additional highly preferred indication is sepsis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Additionally, highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, glioma (e.g., malignant glioma), solid tumors, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic</p>
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45	HHSDI53	162	Production of IL-5	IL-5 FMAT. Assays for immunomodulatory proteins secreted by TH2 cells, mast cells, basophils, and eosinophils that stimulate eosinophil function and B cell Ig production and promote	<p>conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, cardiac reperfusion injury, and asthma and allergy. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").</p> <p>A highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-5 production. An alternative highly preferred embodiment of the invention includes a method for</p>
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				<p>polarization of CD4+ cells into TH2 cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, stimulate immune cell function, modulate B cell Ig production, modulate immune cell polarization, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-5, and the stimulation of eosinophil function and B cell Ig production. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160</p>	<p>stimulating (e.g., increasing) IL-5 production. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) immunoglobulin production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., decreasing) immunoglobulin production. A highly preferred indication includes allergy. A highly preferred indication includes asthma. A highly preferred indication includes rhinitis. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described</p>
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				<p>(2000); Ohshima et al., Blood 92(9):3338-3345 (1998); Jung et al., Eur J Immunol 25(8):2413-2416 (1995); Mori et al., J Allergy Clin Immunol 106(1 Pt 2):558-564 (2000); and Koning et al., Cytokine 9(6):427-436 (1997), the contents of each of which are herein incorporated by reference in its entirety.</p> <p>Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.</p>	<p>below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer.</p> <p>Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, immune reactions to transplanted organs and tissues, hemophilia,</p>
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46	HISBA38	163	Production of IL-10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents	hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease. Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.
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					of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
47	HJPBK28	164	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of	Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis,	

			<p>immunomodulatory genes. Exemplary assays for transcription through the NFkB response element that may be used or routinely modified to test NFkB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic</p>
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48	HKABU43	165	Activation of Adipocyte ERK Signaling Pathway	<p>Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal transduction that regulate cell proliferation or differentiation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK kinase-induced activity of polypeptides of the invention (including</p>	<p>anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.</p> <p>A highly preferred embodiment of the invention includes a method for stimulating adipocyte proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting adipocyte proliferation. A highly preferred embodiment of the invention includes a method for stimulating adipocyte differentiation. An alternative highly preferred embodiment of the invention includes a method for inhibiting adipocyte differentiation. A highly preferred embodiment of the invention includes a method for</p>
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			antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Le Marchand-Brustel Y, Exp Clin Endocrinol Diabetes 107(2):126-132 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Mouse adipocyte cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.	stimulating (e.g., increasing) adipocyte activation. An alternative highly preferred embodiment of the invention includes a method for inhibiting the activation of (e.g., decreasing) and/or inactivating adipocytes. Highly preferred indications include endocrine disorders (e.g., as described below under "Endocrine Disorders"). Highly preferred indications also include neoplastic diseases (e.g., lipomas, liposarcomas, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include blood disorders (e.g., hypertension, congestive heart failure, blood vessel blockage, heart disease, stroke, impotence and/or as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), immune disorders (e.g., as described below under "Immune Activity"), neural disorders (e.g., as described below under "Neural Activity and Neurological Diseases"), and infection (e.g., as described
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					<p>below under "Infectious Disease"). A highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the</p>
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					<p>"Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below (particularly of the urinary tract and skin). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance. Additional highly preferred indications are disorders of the musculoskeletal systems including myopathies, muscular dystrophy, and/or as described herein. Additional highly preferred indications include, hypertension, coronary artery disease, dyslipidemia, gallstones, osteoarthritis, degenerative arthritis, eating disorders, fibrosis, cachexia, and kidney diseases or disorders.</p>
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48	HKABU43	165	Production of IL-10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists	Preferred indications include neoplasms and cancer, such as, lymphoma, leukemia and breast, colon, and kidney cancer. Additional preferred indications include melanoma, prostate, lung, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Highly preferred indications include lipomas and liposarcomas. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.
					Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response,

				<p>or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.</p>	and suppressing a T cell-mediated immune response.
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50	HLYGY91	167	Insulin Secretion	<p>Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and dysregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J, 47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52</p>	<p>A highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyposmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision</p>
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				<p>(1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HIT15 Cells. HIT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.</p>	<p>impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance.</p>
50	HLGY91	167	<p>Production of IL-13 and activation of T-cells.</p>	<p>Assays for production of IL-13 and activation of T-cells are well known in the art and may</p>	<p>Highly preferred indications include allergy and asthma. Additional highly preferred</p>

			<p>be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-13 and/or activation of T-cells. Exemplary assays for IL-13 production that may be used or routinely modified to test activity of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) include, for example, assays such as disclosed and/or cited in: Grunig, G, et al., "Requirement for IL-13 independently of IL-4 in Experimental asthma" Science; 282: 2261-2263 (1998), and Wills-Karp M, et al., "Interleukin-13: central mediator of allergic asthma" Science; 282: 2258-2261 (1998); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL13, a Th2 type cytokine, is a potent</p>	<p>indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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				<p>stimulus for mucus production, airway hyper-responsiveness and allergic asthma. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.</p>	
51	HMC60	168	<p>Activation of transcription through AP1 response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the AP1 response element are known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the AP1 response element that may be used or routinely modified to test AP1-response element activity of polypeptides of the invention (including antibodies</p>	<p>Preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), and infection (e.g., an infectious disease as described below under "Infectious Disease"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as</p>

				<p>and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.</p>	<p>described below) and immunodeficiencies (e.g., as described below). Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include arthritis, asthma, AIDS, allergy, anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, granulomatous disease, inflammatory bowel disease, sepsis, psoriasis,</p>
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51	HMCFH60	168	Activation of transcription through serum response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA	suppression of immune reactions to transplanted organs and tissues, endocarditis, meningitis, and Lyme Disease. A preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) TNF alpha production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) TNF alpha production. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications
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			<p>85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.</p>	<p>include inflammation and inflammatory disorders, and treating joint damage in patients with rheumatoid arthritis. An additional highly preferred indication is sepsis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Additionally, highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, glioma (e.g., malignant glioma), solid tumors, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's</p>
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					lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, cardiac reperfusion injury, and asthma and allergy. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").
51	HMC60	168	Activation of transcription through GAS response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through	Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma (e.g., T cell lymphoma, Burkitt's lymphoma, non-Hodgkins lymphoma, Hodgkin's disease), melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other

			<p>the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the SUPT cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).</p>	<p>preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., viral infections, tuberculosis, infections associated with chronic granulomatous disease and malignant osteoporosis, and/or an infectious disease as described below under "Infectious Disease"). An</p>
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					<p>additional preferred indication is idiopathic pulmonary fibrosis. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, and asthma and allergy.</p>
51	HMCFH60	168	<p>Activation of transcription through STAT6 response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of</p>	<p>A highly preferred indication is allergy. Another highly preferred indication is asthma. Additional highly preferred indications include inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders").</p>

			<p>multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the SUPT cell line, which is a suspension</p>	<p>Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous</p>
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				<p>culture of IL-2 and IL-4 responsive T cells.</p>	<p>disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").</p>
52	HMI AK10	169	<p>Activation of transcription through cAMP response element (CRE) in pre-adipocytes.</p>	<p>Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway.</p>	<p>A highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. An additional highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy,</p>

				<p>CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may</p>	<p>nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hypermolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). Additional highly preferred indications are</p>
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52	HMI AK10	169	<p>Activation of transcription through STAT6 response element in immune cells (such as mast cells).</p>	<p>be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.</p> <p>Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element in immune cells (such as in the human HMC-1 mast cell line) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the</p>	<p>complications associated with insulin resistance.</p>
				<p>Highly preferred indications include allergy, asthma, and rhinitis. Additional highly preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications also include hematopoietic and immunological disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below).</p>	

				<p>polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Sherman, Immunol Rev 179:48-56 (2001); Malaviya and Uckun, J Immunol 168:421-426 (2002); Masuda et al., J Biol Chem 275(38):29331-29337 (2000); and Masuda et al., J Biol Chem 276:26107-26113 (2001), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.</p>	<p>Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancer, such as, for example, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include hematopoietic and immunological disorders such as arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
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52	HMI AK10	169	<p>Activation of transcription through serum response element in immune cells (such as natural killer cells).</p>	<p>Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of</p>	<p>A preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) TNF alpha production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) TNF alpha production. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders, and treating joint damage in patients with rheumatoid arthritis. An additional highly preferred</p>
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				<p>which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.</p>	<p>indication is sepsis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Additionally, highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, glioma (e.g., malignant glioma), solid tumors, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, neutropenia, neutrophilia, psoriasis, suppression of</p>
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52	HMIAK10	169	<p>Activation of transcription through GAS response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including</p>	<p>immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, cardiac reperfusion injury, and asthma and allergy. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").</p> <p>Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders").</p> <p>Highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma (e.g., T cell lymphoma, Burkitt's lymphoma, non-Hodgkins lymphoma, Hodgkin's disease), melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or</p>
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			<p>antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the SUPT cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).</p>	<p>dysplasia. Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional preferred indications include inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., viral infections, tuberculosis, infections associated with chronic granulomatous disease and malignant osteoporosis, and/or an infectious disease as described below under "Infectious Disease"). An additional preferred indication is idiopathic pulmonary fibrosis. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia,</p>
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					acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, and asthma and allergy.
52	HMI AK10	169	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element	Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is

				<p>activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia,</p>
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52	HMIK10	169	<p>Activation of transcription through STAT6 response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and</p>	<p>neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.</p> <p>A highly preferred indication is allergy. Another highly preferred indication is asthma. Additional highly preferred indications include inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative</p>
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				<p>Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>Disorders"). Preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.</p> <p>Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease. An additional preferred indication is infection (e.g., an infectious disease as described</p>
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53	HMIBD93	170	Production of IL-13 and activation of T-cells.	Assays for production of IL-13 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-13 and/or activation of T-cells. Exemplary assays for IL-13 production that may be used or routinely modified to test activity of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) include, for example, assays such as disclosed and/or cited in: Grunig, G, et al., "Requirement for IL-13 independently of IL-4 in Experimental asthma" Science; 282: 2261-2263 (1998), and Wills-Karp M, et al., "Interleukin-13: central mediator of allergic asthma" Science; 282: 2258-2261 (1998); the contents of each of	below under "Infectious Disease"). Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.
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					which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL13, a Th2 type cytokine, is a potent stimulus for mucus production, airway hyper-responsiveness and allergic asthma. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
54	HMWJF53	171	Inhibition of squalene synthetase gene transcription.		Reporter Assay: construct contains regulatory and coding sequence of squalene synthetase, the first specific enzyme in the cholesterol biosynthetic pathway. See Jiang, et al., J. Biol. Chem. 268:12818-12824(1993), the contents of which are herein incorporated by reference in its entirety. Cells were treated with	

				SID supernatants, and SEAP activity was measured after 72 hours. HepG2 is a human hepatocellular carcinoma cell line (ATCC HB-8065). See Knowles et al., Science. 209:497-9 (1980), the contents of which are herein incorporated by reference in its entirety.	
54	HMWJF53	171	Production of IL-5	IL-5 FMAT. Assays for immunomodulatory proteins secreted by TH2 cells, mast cells, basophils, and eosinophils that stimulate eosinophil function and B cell Ig production and promote polarization of CD4+ cells into TH2 cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, stimulate immune cell function, modulate B cell Ig production, modulate immune cell polarization, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins	A highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-5 production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-5 production. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) immunoglobulin production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., decreasing) immunoglobulin production. A highly preferred indication includes allergy. A highly preferred indication includes asthma. A highly preferred indication includes rhinitis.

			<p>evaluate the production of cytokines, such as IL-5, and the stimulation of eosinophil function and B cell Ig production. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Ohshima et al., Blood 92(9):3338-3345 (1998); Jung et al., Eur J Immunol 25(8):2413-2416 (1995); Mori et al., J Allergy Clin Immunol 106(1 Pt 2):558-564 (2000); and Koning et al., Cytokine 9(6):427-436 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human</p>	<p>An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative</p>
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				lymphocytes that mature in the thymus and express a T cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.	disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.
55	HNECL22	172	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of	Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis,

				<p>immunomodulatory genes. Exemplary assays for transcription through the NFkB response element that may be used or routinely modified to test NFkB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic</p>
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56	HNFAC50	173	Regulation of apoptosis of immune cells (such as mast cells).	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate caspase protease-mediated apoptosis in immune cells (such as, for example, in mast cells). Mast cells are found in connective and mucosal tissues throughout the body, and their activation via immunoglobulin E -antigen, promoted by T helper cell type 2 cytokines, is an important component of allergic disease.	anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.	Preferred embodiments of the invention include using polypeptides of the invention (or antibodies, agonists, or antagonists thereof) in detection, diagnosis, prevention, and/or treatment of asthma, allergy, hypersensitivity and inflammation.
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				<p>Dysregulation of mast cell apoptosis may play a role in allergic disease and mast cell tumor survival. Exemplary assays for caspase apoptosis that may be used or routinely modified to test caspase apoptosis activity induced by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Masuda A, et al., <i>J Biol Chem</i>, 276(28):26107-26113 (2001); Yeatman CF 2nd, et al., <i>J Exp Med</i>, 192(8):1093-1103 (2000); Lee et al., <i>FEBS Lett</i> 485(2-3): 122-126 (2000); Nor et al., <i>J Vasc Res</i> 37(3): 209-218 (2000); and Karsan and Harlan, <i>J Atheroscler Thromb</i> 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Immune cells that may be used according to these assays are publicly available (e.g., through commercial sources). Exemplary immune cells that may be used according to these assays include mast cells such as the HMC human mast cell line.</p>	
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57	HNGEA34	174	Production of IL-5	<p>IL-5 FMAT. Assays for immunomodulatory proteins secreted by TH2 cells, mast cells, basophils, and eosinophils that stimulate eosinophil function and B cell Ig production and promote polarization of CD4+ cells into TH2 cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, stimulate immune cell function, modulate B cell Ig production, modulate immune cell polarization, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-5, and the stimulation of eosinophil function and B cell Ig production. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and</p>	<p>A highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-5 production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-5 production. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) immunoglobulin production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., decreasing) immunoglobulin production. A highly preferred indication includes allergy. A highly preferred indication includes asthma. A highly preferred indication includes rhinitis. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related</p>
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			<p>agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Ohshima et al., Blood 92(9):3338-3345 (1998); Jung et al., Eur J Immunol 25(8):2413-2416 (1995); Mori et al., J Allergy Clin Immunol 106(1 Pt 2):558-564 (2000); and Koning et al., Cytokine 9(6):427-436 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.</p>	<p>Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple</p>
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58	HNGIV64	175	Activation of Adipocyte ERK Signaling Pathway	<p>Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal transduction that regulate cell proliferation or differentiation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in</p>	<p>myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p> <p>A highly preferred embodiment of the invention includes a method for stimulating adipocyte proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting adipocyte proliferation. A highly preferred embodiment of the invention includes a method for stimulating adipocyte differentiation. An alternative highly preferred embodiment of the invention includes a method for inhibiting adipocyte differentiation. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) adipocyte activation. An alternative highly preferred</p>
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			<p>Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Le Marchand-Brustel Y, Exp Clin Endocrinol Diabetes 107(2):126-132 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Mouse adipocyte cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.</p>	<p>embodiment of the invention includes a method for inhibiting the activation of (e.g., decreasing) and/or inactivating adipocytes. Highly preferred indications include endocrine disorders (e.g., as described below under "Endocrine Disorders"). Highly preferred indications also include neoplastic diseases (e.g., lipomas, liposarcomas, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include blood disorders (e.g., hypertension, congestive heart failure, blood vessel blockage, heart disease, stroke, impotence and/or as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), immune disorders (e.g., as described below under "Immune Activity"), neural disorders (e.g., as described below under "Neural Activity and Neurological Diseases"), and infection (e.g., as described below under "Infectious Disease"). A highly preferred indication is diabetes</p>
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					<p>mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic</p>
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					<p>retinopathy and blindness), ulcers and impaired wound healing, infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below (particularly of the urinary tract and skin). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance. Additional highly preferred indications are disorders of the musculoskeletal systems including myopathies, muscular dystrophy, and/or as described herein. Additional highly preferred indications include, hypertension, coronary artery disease, dyslipidemia, gallstones, osteoarthritis, degenerative arthritis, eating disorders, fibrosis, cachexia, and kidney diseases or disorders. Preferred indications include neoplasms and cancer, such as, lymphoma, leukemia and breast,</p>
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					<p>colon, and kidney cancer.</p> <p>Additional preferred indications include melanoma, prostate, lung, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Highly preferred indications include lipomas and liposarcomas. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.</p>
58	HNGIV64	175	<p>Production of IL-10 and activation of T-cells.</p>	<p>Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation</p>	<p>Highly preferred indications include allergy and asthma.</p> <p>Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>

				include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	

59	HNGKT41	176	<p>Activation of transcription through cAMP response element (CRE) in pre-adipocytes.</p>	<p>Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays</p>	<p>A highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. An additional highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as</p>
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				disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.	described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). Additional highly preferred indications are complications associated with insulin resistance.
59	HNGKT41	176	Activation of transcription through NFKB response	This reporter assay measures activation of the NFKB signaling pathway in Ku812 human	Highly preferred indication includes allergy, asthma, and rhinitis. Additional highly

			<p>basophil cell line. Assays for the activation of transcription through the NFkB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFkB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFkB response element that may be used or routinely modified to test NFkB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Marone et al, Int Arch Allergy Immunol 114(3):207-17 (1997), the contents of each of which are herein incorporated by reference in its entirety. Basophils that</p>	<p>preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications include immunological and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"). Preferred indications also include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancer, such as, for example, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, urinary tract cancers and as described below under</p>
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				may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human basophil cell lines that may be used according to these assays include Ku812, originally established from a patient with chronic myelogenous leukemia. It is an immature prebasophilic cell line that can be induced to differentiate into mature basophils.	"Hyperproliferative Disorders".
59	HNGKT41	176	Activation of transcription through serum response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of	A preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) TNF alpha production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) TNF alpha production. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus,

				<p>the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.</p>	<p>Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders, and treating joint damage in patients with rheumatoid arthritis. An additional highly preferred indication is sepsis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Additionally, highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, glioma (e.g., malignant glioma), solid tumors, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic</p>
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					<p>conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, cardiac reperfusion injury, and asthma and allergy. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").</p>
59	HNGKT41	176	<p>Activation of transcription through API response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the API response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention</p>	<p>Preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as described below under "Immune Activity", "Cardiovascular</p>

				<p>(including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the API response element that may be used or routinely modified to test API-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., <i>Gene</i> 66:1-10 (1988); Cullen and Malm, <i>Methods in Enzymol</i> 216:362-368 (1992); Henthorn et al., <i>Proc Natl Acad Sci USA</i> 85:6342-6346 (1988); Rellahan et al., <i>J Biol Chem</i> 272(49):30806-30811 (1997); Chang et al., <i>Mol Cell Biol</i> 18(9):4986-4993 (1998); and Fraser et al., <i>Eur J Immunol</i> 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according</p>	<p>Disorders”, and/or “Blood-Related Disorders”), and infection (e.g., an infectious disease as described below under “Infectious Disease”). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under “Hyperproliferative Disorders”). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred</p>
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				to these assays include the SUPT cell line, which is an IL-2 and IL-4 responsive suspension-culture cell line.	indications include arthritis, asthma, AIDS, allergy, anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, granulomatous disease, inflammatory bowel disease, sepsis, psoriasis, suppression of immune reactions to transplanted organs and tissues, endocarditis, meningitis, and Lyme Disease.
59	HNGKT41	176	Activation of transcription through CD28 response element in immune cells (such as T-cells).	Assays for the activation of transcription through the CD28 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T cells. Exemplary assays for transcription through the CD28 response element that may be used or routinely modified to test CD28-response element activity of polypeptides of the invention (including antibodies	<p>A highly preferred embodiment of the invention includes a method for stimulating T cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting T cell proliferation. A highly preferred embodiment of the invention includes a method for activating T cells. An alternative highly preferred embodiment of the invention includes a method for inhibiting the activation of and/or inactivating T cells. A highly preferred embodiment of the invention includes a method for stimulating (e.g.,</p>

				<p>and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol 159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>increasing) IL-2 production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-2 production. Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Highly preferred indications include neoplastic diseases (e.g., melanoma, renal cell carcinoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, melanoma (e.g., metastatic melanoma), renal cell carcinoma (e.g., metastatic renal cell carcinoma), leukemia, lymphoma (e.g., T cell lymphoma), and prostate,</p>
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					breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. A highly preferred indication includes infection (e.g., AIDS, tuberculosis, infections associated with granulomatous disease, and osteoporosis, and/or as described below under "Infectious Disease"). A highly preferred indication is AIDS. Additional highly preferred indications include suppression of immune reactions to transplanted organs and/or tissues, uveitis, psoriasis, and tropical spastic paraparesis. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute
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					lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.
59	HNGKT4I	176	Activation of transcription through NFAT response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies	Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. An additional highly

			<p>and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis,</p>
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59	HNGKT41	176	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol	suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy. Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as
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				<p>216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.</p>
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60	HNGNO53	177	Inhibition of squalene synthetase gene transcription.	<p>Reporter Assay: construct contains regulatory and coding sequence of squalene synthetase, the first specific enzyme in the cholesterol biosynthetic pathway. See Jiang, et al., J. Biol. Chem. 268:12818-12824(1993), the contents of which are herein incorporated by reference in its entirety. Cells were treated with SID supernatants, and SEAP activity was measured after 72 hours. HepG2 is a human hepatocellular carcinoma cell line (ATCC HB-8065). See Knowles et al., Science. 209:497-9 (1980), the contents of which are herein incorporated by reference in its entirety.</p>	
60	HNGNO53	177	Activation of transcription through serum response element in immune cells (such as T-cells).	<p>Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved</p> <p>A preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) TNF alpha production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) TNF alpha production. Preferred indications include blood disorders (e.g., as described below under "Immune</p>	

			<p>in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.</p>	<p>Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders, and treating joint damage in patients with rheumatoid arthritis. An additional highly preferred indication is sepsis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Additionally, highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, melanoma, glioma (e.g., malignant glioma), solid tumors, and prostate, breast,</p>
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	HNGNO53	177	Production of IL-10	Assays for production of IL-10	<p>lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, cardiac reperfusion injury, and asthma and allergy. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").</p> <p>Highly preferred indications</p>
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60			and activation of T-cells.	<p>and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells.</p>	<p>include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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61	HNHCT47	178	<p>Regulation of viability or proliferation of immune cells (such as human eosinophil EOL-1 cells).</p>	<p>IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.</p>	<p>Highly preferred indications include eosinophilia, asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as</p>
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				<p>viable cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells.</p> <p>Eosinophils are a type of immune cell important in allergic responses; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction.</p> <p>Eosinophil cell lines that may be used according to these assays are publicly available and/or may be routinely generated.</p> <p>Exemplary eosinophil cells that may be used according to these assays include EOL-1 Cells.</p>	<p>described below). Highly preferred indications also include boosting or inhibiting immune cell proliferation.</p> <p>Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders").</p> <p>Highly preferred indications include boosting an eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.</p>
61	HNHCT47	178	<p>Production of IL-10 and activation of T-cells.</p>	<p>Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells.</p> <p>Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of</p>	<p>Highly preferred indications include allergy and asthma.</p> <p>Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T</p>

				<p>the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes</p>	cell-mediated immune response, and suppressing a T cell-mediated immune response.
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62	HNHK174	179	Production of VCAM in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))	isolated from cord blood. Assays for measuring expression of VCAM are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate VCAM expression. For example, FMAT may be used to measure the upregulation of cell surface VCAM-1 expression in endothelial cells. Endothelial cells are cells that line blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are available from commercial sources. The expression of VCAM (CD106), a membrane-associated protein, can be upregulated by cytokines or other factors, and contributes	Highly preferred indications include inflammation (acute and chronic), restenosis, atherosclerosis, asthma and allergy. Highly preferred indications include inflammation and inflammatory disorders, immunological disorders, neoplastic disorders (e.g. cancer/tumorigenesis), and cardiovascular disorders (such as described below under "Immune Activity", "Blood-Related Disorders", "Hyperproliferative Disorders" and/or "Cardiovascular Disorders"). Highly preferred indications include neoplasms and cancers such as, for example, leukemia, lymphoma, melanoma, renal cell carcinoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.
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				to the extravasation of lymphocytes, leucocytes and other immune cells from blood vessels; thus VCAM expression plays a role in promoting immune and inflammatory responses.	
62	HNHK174	179	Production of IL-10 and activation of T-cells.	<p>Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells.</p> <p>Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-</p>	<p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>

				helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
63	HORBS82	180	Activation of Adipocyte ERK Signaling Pathway	Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal transduction that regulate cell proliferation or differentiation are well known in the art and may be used or routinely modified to assess the ability of A highly preferred embodiment of the invention includes a method for stimulating adipocyte proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting adipocyte	

			<p>polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Le Marchand-Brustel Y, Exp Clin Endocrinol Diabetes 107(2):126-132 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Mouse adipocyte cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse adipocyte cells that may be used</p>	<p>proliferation. A highly preferred embodiment of the invention includes a method for stimulating adipocyte differentiation. An alternative highly preferred embodiment of the invention includes a method for inhibiting adipocyte differentiation. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) adipocyte activation. An alternative highly preferred embodiment of the invention includes a method for inhibiting the activation of (e.g., decreasing) and/or inactivating adipocytes. Highly preferred indications include endocrine disorders (e.g., as described below under "Endocrine Disorders"). Highly preferred indications also include neoplastic diseases (e.g., lipomas, liposarcomas, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include blood disorders (e.g., hypertension, congestive heart failure, blood vessel blockage, heart disease, stroke, impotence</p>
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				<p>according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.</p> <p>and/or as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders", immune disorders (e.g., as described below under "Immune Activity"), neural disorders (e.g., as described below under "Neural Activity and Neurological Diseases"), and infection (e.g., as described below under "Infectious Disease"). A highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness,</p>
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					<p>nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below (particularly of the urinary tract and skin). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance. Additional highly preferred indications are</p>
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63	HORBS82	180	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be	Highly preferred indications include asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly	disorders of the musculoskeletal systems including myopathies, muscular dystrophy, and/or as described herein. Additional highly preferred indications include, hypertension, coronary artery disease, dyslipidemia, gallstones, osteoarthritis, degenerative arthritis, eating disorders, fibrosis, cachexia, and kidney diseases or disorders. Preferred indications include neoplasms and cancer, such as, lymphoma, leukemia and breast, colon, and kidney cancer. Additional preferred indications include melanoma, prostate, lung, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Highly preferred indications include lipomas and liposarcomas. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.
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				<p>used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions;</p>	<p>preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below). Highly preferred indications also include boosting or inhibiting immune cell proliferation. Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.</p>
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				<p>they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosphorylation of JUN N-terminal kinase and</p>
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				<p>failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.</p>	
63	HORBS82	180	<p>Regulation of apoptosis of immune cells (such as mast cells).</p>	<p>Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate caspase protease-mediated apoptosis in immune cells (such as, for example, in mast cells). Mast cells are found in connective and mucosal tissues throughout the body, and their activation via immunoglobulin E -antigen, promoted by T helper cell type 2 cytokines, is an important component of allergic disease. Dysregulation of mast cell apoptosis may play a role in allergic disease and mast cell tumor survival. Exemplary assays for caspase apoptosis that may be used or routinely</p>	<p>Preferred embodiments of the invention include using polypeptides of the invention (or antibodies, agonists, or antagonists thereof) in detection, diagnosis, prevention, and/or treatment of asthma, allergy, hypersensitivity and inflammation.</p>

				<p>modified to test caspase apoptosis activity induced by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Masuda A, et al., J Biol Chem, 276(28):26107-26113 (2001); Yeatman CF 2nd, et al., J Exp Med, 192(8):1093-1103 (2000); Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Immune cells that may be used according to these assays are publicly available (e.g., through commercial sources). Exemplary immune cells that may be used according to these assays include mast cells such as the HMC human mast cell line.</p>	
63	HORBS82	180	<p>Production of IL-13 and activation of T-cells.</p>	<p>Assays for production of IL-13 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of</p>	<p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as</p>

				<p>polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-13 and/or activation of T-cells. Exemplary assays for IL-13 production that may be used or routinely modified to test activity of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) include, for example, assays such as disclosed and/or cited in: Grunig, G, et al., "Requirement for IL-13 independently of IL-4 in Experimental asthma" Science; 282: 2261-2263 (1998), and Wills-Karp M, et al., "Interleukin-13: central mediator of allergic asthma" Science; 282: 2258-2261 (1998); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL13, a Th2 type cytokine, is a potent stimulus for mucus production, airway hyper-responsiveness</p>	<p>described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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64	HOUDE92	181	<p>and allergic asthma. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.</p> <p>Assays for the activation of transcription through the API response element are known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the API response element that may be used or routinely modified to test API-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays</p>	<p>Preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), and infection (e.g., an infectious disease as described below under "Infectious Disease"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as</p>
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				<p>disclosed in Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.</p>	<p>described below). Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include arthritis, asthma, AIDS, allergy, anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, granulomatous disease, inflammatory bowel disease, sepsis, psoriasis, suppression of immune reactions to transplanted organs</p>
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64	HOUDE92	181	<p>Activation of transcription through cAMP response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP and regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Genes 15(2):105-117</p>	<p>and tissues, endocarditis, meningitis, and Lyme Disease.</p> <p>Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional preferred indications include inflammation and inflammatory disorders. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for</p>
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64	transcription through GATA-3 response element in immune cells (such as mast cells).	activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol	include allergy, asthma, and rhinitis. Additional preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications also include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary tract cancers and/or as described below under "Hyperproliferative Disorders"). Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example,
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				<p>216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.</p>	<p>hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
64	HOUDE92	181	<p>Activation of transcription through NFAT response element in immune cells (such as mast</p>	<p>This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been</p>	<p>Highly preferred indications include allergy, asthma, and rhinitis. Additional preferred indications include infection (e.g., an infectious disease as</p>

			<p>linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et</p>	<p>described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications also include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary tract cancers and/or as described below under "Hyperproliferative Disorders"). Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia,</p>
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				<p>al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.</p>	<p>leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
64	HOUDE92	181	<p>Activation of transcription through NFAT response in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the</p>	<p>Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus,</p>

				<p>invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the</p>	<p>multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred</p>
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64	HOUDE92	181	<p>Activation of transcription through serum response element in immune cells (such as natural killer cells).</p>	<p>Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the function of growth-related genes</p>	<p>ATCC). Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.</p>	<p>indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.</p> <p>A preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) TNF alpha production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) TNF alpha production. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or</p>
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				<p>in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.</p>	<p>"Cardiovascular Disorders"), Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders, and treating joint damage in patients with rheumatoid arthritis. An additional highly preferred indication is sepsis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Additionally, highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, glioma (e.g., malignant glioma), solid tumors, and prostate, breast, lung, colon, pancreatic,</p>
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					<p>esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, cardiac reperfusion injury, and asthma and allergy. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").</p>
					Assays for the activation of transcription through the NFkB
					Activation of transcription through
					181
					HOUDE92
64					Preferred embodiments of the invention include using

			<p>NFKB response element in immune cells (such as B-cells).</p>	<p>response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Gri G, et al., Biol Chem, 273(1):6431-6438 (1998); Pyatt DW, et al., Cell Biol Toxicol 2000;16(1):41-51 (2000); Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Valle Blazquez et al, Immunology 90(3):455-460 (1997); Aramburau et al., J Exp Med</p>	<p>polypeptides of the invention (or antibodies, agonists, or antagonists thereof) in detection, diagnosis, prevention, and/or treatment of Cancer, Autoimmunity, Allergy and Asthma</p>
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64	HOUDE92	181	<p>Activation of transcription through NFKB response element in neuronal cells (such as SKNMC cells).</p>	<p>82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Immune cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary immune cells that may be used according to these assays include the Reh B-cell line.</p>	<p>Preferred embodiments of the invention include using polypeptides of the invention (or antibodies, agonists, or antagonists thereof) in detection, diagnosis, prevention, and/or treatment of Neurological Diseases and Disorders (e.g. Alzheimer's Disease, Parkinson's Disease, Brain Cancer, Seizures).</p>
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				<p>the invention) include assays disclosed in: Gill JS, et al., Neurobiol Dis, 7(4):448-461 (2000); Tamatani M, et al., J Biol Chem, 274(13):8531-8538 (1999); Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Valle Blazquez et al, Immunology 90(3):455-460 (1997); Aramburau et al., J Exp Med 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Neuronal cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary neuronal cells that may be used according to these assays include the SKNMC neuronal cell line.</p>	<p>Preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as</p>
64	HOUDE92	181	<p>Activation of transcription through AP1 response element in immune cells (such as T-</p>	<p>Assays for the activation of transcription through the AP1 response element are well-known in the art and may be used or routinely modified to</p>	

			cells).	<p>assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the API response element that may be used or routinely modified to test API-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays are publicly available (e.g., through the</p>	<p>described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), and infection (e.g., an infectious disease as described below under "Infectious Disease"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example,</p>
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				ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is an IL-2 and IL-4 responsive suspension-culture cell line.	hyperplasia, metaplasia, and/or dysplasia. Preferred indications include arthritis, asthma, AIDS, allergy, anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, granulomatous disease, inflammatory bowel disease, sepsis, psoriasis, suppression of immune reactions to transplanted organs and tissues, endocarditis, meningitis, and Lyme Disease.
64	HOUDE92	181	Activation of transcription through CD28 response element in immune cells (such as T-cells).	Assays for the activation of transcription through the CD28 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T cells. Exemplary assays for transcription through the CD28 response element that may be used or routinely modified to test CD28-response element	<p>A highly preferred embodiment of the invention includes a method for stimulating T cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting T cell proliferation. A highly preferred embodiment of the invention includes a method for activating T cells. An alternative highly preferred embodiment of the invention includes the activation of and/or inactivating T cells. A highly preferred embodiment</p>

				<p>activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol 159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>of the invention includes a method for stimulating (e.g., increasing) IL-2 production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-2 production. Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Highly preferred indications include neoplastic diseases (e.g., melanoma, renal cell carcinoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, melanoma (e.g., metastatic melanoma), renal cell carcinoma (e.g., metastatic renal cell carcinoma),</p>
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					<p>leukemia, lymphoma (e.g., T cell lymphoma), and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. A highly preferred indication includes infection (e.g., AIDS, tuberculosis, infections associated with granulomatous disease, and osteoporosis, and/or as described below under "Infectious Disease"). A highly preferred indication is AIDS. Additional highly preferred indications include suppression of immune reactions to transplanted organs and/or tissues, uveitis, psoriasis, and tropical spastic paraparesis. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications also include anemia, pancytopenia,</p>
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64	HOUDE92	181	Activation of transcription through NFAT response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element	leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include
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			<p>activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>inflammation and inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease,</p>
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64	HOUDE92	181	<p>Activation of transcription through NFKB response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene</p>	<p>sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.</p> <p>Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic</p>
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				<p>66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune</p>
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					reactions to transplanted organs, asthma and allergy.
64	HOUDE92	181	<p>Activation of transcription through STAT6 response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538</p>	<p>A highly preferred indication is allergy. Another highly preferred indication is asthma. Additional highly preferred indications include inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon,</p>

				<p>(1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").</p>
65	HOUFS04	182	Activation of transcription through	Assays for the activation of transcription through the	Highly preferred indications include neoplastic diseases (e.g.,

				<p>GAS response element in immune cells (such as T-cells).</p>	<p>Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Hentinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are</p>	<p>leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma (e.g., T cell lymphoma, Burkitt's lymphoma, non-Hodgkins lymphoma, Hodgkin's disease), melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional preferred indications include inflammation and inflammatory disorders.</p>
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